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ACUTE PANCREATITIS.[1]

By BALCOMBE QUICK.

[From the Alfred Hospital, Melbourne.]

THERE have been occasions in the experience of most surgeons when the escape of blood-stained fluid on incision of the peritoneum, followed by the demonstration of areas of necrosis in the omental fat, has at once made clear the nature of an illness which had been of some obscurity.

The reason for this is not far to seek. In the first place, acute pancreatic disease is so very generally held to be one of the rarest causes of an abdominal crisis that the very existence of the pancreas is apt to be forgotten when the diagnosis is under consideration, or, secondly, the clinical picture may present enough superficial resemblance to other acute abdominal conditions to divert attention from the true seat of the illness.

An attempt is made in the present paper to correct the somewhat erroneous impression of extreme rarity, to clarify the diagnosis and to discuss the principles of treatment.

INCIDENCE.

The incidence is probably considerably higher than is generally believed. Chamberlain,[1] in reporting 21 cases from the General Infirmary, Leeds, which had arisen during the period of ten years, 1915 to 1924, states that acute pancreatitis occurred only once in every 5,000 surgical admissions. McNeill Love[2] reported 51 cases from the London Hospital between the years 1911 and 1924. Schmieden and Sebening,[3] from their own clinical material and from that of approximately one hundred other German clinics, obtained records of 1,510 patients, of whom 1,278 were submitted to operation. These cases occurred during a period of

[1] Read at the annual meeting of the Royal Australasian College of Surgeons, February, 1932.

eight years, ending in 1927. They believe that the disease is becoming more commonly seen and that the apparently increasing frequency, in large part due, no doubt, to earlier intervention in all acute abdominal affections, is perhaps to an extent real, contrasting as it does with a very definite decline in frequency during the lean years of the War.

During the past four years forty-five proved cases have occurred in patients admitted into the Alfred Hospital, Melbourne, representing one case in every 578 admissions, medical as well as surgical. It may be mentioned, for purposes of comparison, that during the same period there were admitted 65 patients with perforated duodenal or gastric ulcer.

It is interesting to find that this ratio roughly approximates to that given by Schmieden and Sebening from the Frankfurt Clinic—38 cases of pancreatitis and 63 of perforation. Of these 45 patients, 17 were under my own care and all have been closely watched and followed, particularly from the aspect of diagnosis, by Dr. Mushin, the Medical Superintendent, whose paper follows. A study of this series and of four other cases operated upon by myself outside the hospital during the same period furnishes the basis of the present paper.

Some hesitation has been felt in the use of the title "Acute Pancreatitis", for the term denotes inflammation and it may be further inferred that such inflammation must be of microbic origin. The immediate cause of the primary pancreatic lesions, œdema and cellular necrosis, is in all probability a cytolysis of the gland tissue, not necessarily associated with bacterial activities. A continuation of this process, associated with the liberation of activated ferments, is responsible for the capillary destruction, hæmorrhages and fat necrosis which are such outstanding features of the disease. The belief that the lesions are of biochemical as opposed to inflammatory origin has led several recent writers to choose the term "acute pancreatic necrosis" in their papers upon the subject. Provided it be clearly recognized that the necrosis is by no means constantly gross and macroscopic, this entitlement is to be commended. Nevertheless, the older term carries with it the sanction of long usage and it has been thought well to retain it.

ÆTIOLOGICAL CONSIDERATIONS.

Although it is outside the scope of the present paper to deal at length with theories of causation, short reference to them is necessary on account of their intimate bearing upon treatment. The view that the primary lesion, necrosis of the pancreatic cells, is due to activation of ferments *in situ* is very generally held. The division in opinion occurs between those who follow the teaching of Maugeret, Deaver and Mann that the cellular and other products of lymph-borne infection constitute the activating agents, and those others, the majority, who believe that the process is one in which some mechanical defect (blockage) or physiological error (spasm) at the biliary outlet brings about a reflux of bile into the pancreatic duct. This view may be referred to as the

"canalicular" theory of origin. Archibald[4] holds, on experimental and microscopic evidence, that the primary cellular necrosis and accompanying œdema are due to a direct chemically destructive action of bile, a cytolysis, followed perhaps by the liberation of activating substances and an autolysis which causes the grosser manifestations of necrosis. This distinction appears to be of some importance as supplying an explanation of the lighter manifestations of pancreatic involvement, of which mention will be made later.

Since Opie's classical observation in 1901 that impaction of a gall stone at the papilla of Vater might canalize (throw into a continuous channel) the common duct and duct of Wirsung, this method of production has probably been the most widely accepted. Certain physical conditions—suitable size and shape of the calculus and a particular anatomical arrangement at the union of the ducts—must be fulfilled in order that such canalization may occur. Many other cases similar to Opie's have subsequently been reported, and it would appear at first sight that this physical explanation of activation of bile invasion was entirely satisfactory.

Unfortunately for the easy acceptance of this attractive theory, Mann found that the necessary anatomical arrangement existed in only 3·5% of the 200 cadavers he examined. He has shown, too, that it is not possible or, at any rate, not easy to produce experimental pancreatitis with any degree of certainty in this way, using sterile bile and such conditions of bile pressure as might obtain normally in the body. Archibald showed, however, that the injection of infected bile, or bile deprived of its mucus, or in which the salts were concentrated, was almost constantly successful. It will be noted that any or all of these conditions may be present in disease of the gall bladder.

A further difficulty lies in the fact that only in a minority of cases of pancreatitis is a stone found in the ampulla at operation or autopsy. A stone was found in 18% of Schmieden and Sebening's personal cases, and in 4·5% of those they collected. Thirty *per centum* of the cases showed no sign of gall stones whatever. But by the same showing it is evident that there was an associated cholelithiasis in 70% of cases in their series: and in a considerable proportion of this 70% the stones were found in the common duct.

In an attempt to explain the occurrence of pancreatitis in those cases unassociated with gall stones, Archibald[4] developed the theory of a failure of relaxation or spasm of the sphincter of Oddi. He carried out numerous experiments, using cats, in which the tonus of the sphincter constituted the sole obstacle to flow into the duodenum. The tonus, he found, would resist a pressure of 450 to 600 millimetres of bile attained by means of funnel and tubing with a cannula tied into the gall bladder. A rapidly fatal pancreatitis was almost uniformly produced with infected bile: in one instance death resulted within twenty minutes, a complete *post mortem* picture of necrosis and hæmorrhage being found. When mucus-free bile or concentrated solutions of bile

salts were used non-fatal pancreatic necrosis very constantly resulted. Archibald, indeed, has gone so far as to state that in his belief bile reflux is in all cases, even those in which stones are found in the common duct, due to spasm of the sphincter.

The theory of infective activation as upheld by Deaver.[5] Mann and others is difficult to substantiate experimentally. They believe that an inflammatory process spreads to the pancreas by lymphatic channels from some focus in an adjacent viscus, be it gall bladder, duodenum or stomach. But it has not been found possible to produce lesions resembling pancreatitis by infecting the liver or gall bladder with staphylococci, although the organisms may be traced from the gall bladder to the cystic gland and thence along the common duct to the head of the pancreas.

Clinically, too, it is impossible to reconcile the frequency of infective conditions of the gall bladder and of penetrating gastric and duodenal ulcer with the comparative rarity of pancreatitis and its extreme rarity in association with ulcer. Moreover, in many fatal cases of pancreatitis both peritoneal exudate and bile are found to be sterile. In view of Archibald's experimental findings, that only by the use of infected bile could he produce a certainly fatal lesion, it must be admitted that this last observation is somewhat difficult of explanation under either theory of causation.

Bacteriological studies were made in only nine cases of the series. It will be evident at once (see Table I) that the results obtained are equivocal. High-grade lesions, leading to a fatal issue, are found associated with sterile bile and (accidental contamination of the) peritoneal fluid, whilst recovery has followed in spite of evidence of gross infection, the rôle of which in determining either the occurrence of the disease, its type or its outcome is thus by no means clear.

The balance of experimental evidence, however, appears to favour the canalicular theory of origin rather than the lymph-borne infection

TABLE I.

Case.	Type of Lesion.	Duration.	Result.	Bacteriological Report.
I	Acute pancreatic œdema.	17 hours.	Recovery.	No growth.
II	Acute pancreatic œdema.	36 hours.	Recovery.	Two types of coliform organisms from both gall bladder and common duct bile.
III	Acute hæmorrhagic pancreatitis.	12 hours.	Death (1 week).	Peritoneal fluid: *Bacillus coli* and streptococci.
IV	Acute hæmorrhagic pancreatitis.	12 hours.	Death (on table).	Gall bladder wall and bile sterile. Peritoneal fluid *Staphylococcus albus* (probable contamination).
V	Acute hæmorrhagic pancreatitis.	17 hours.	Recovery.	No growth from pile or peritoneal fluid.
VI	Acute hæmorrhagic pancreatitis.	48 hours.	Death (5 weeks).	No growth common duct bile. Thick black gall bladder bile grew *Bacillus alkaligenes*.
VII	Acute hæmorrhagic pancreatitis.	48 hours.	Recovery.	Common duct bile grew a proteus and a Gram-positive hæmolytic bacillus, non-sporing anaerobic rather than aerobic.
VIII	Acute hæmorrhagic pancreatitis.	36 hours.	Recovery.	Peritoneal fluid: *Bacillus welchii*.
IX	Acute hæmorrhagic pancreatitis.	72 hours.	Death (3 days).	Bile: no growth.

of Deaver, and I shall later offer some clinical observations made in certain cases of the series in its further support.

Two striking facts emerge from a confusing mass of clinical and laboratory data:

(i) The high incidence of associated cholelithiasis (50% to 70%, various reports; 61% in the present series).

(ii) The ease of production of experimental pancreatic necrosis, either following the forcible injection of sterile normal bile into the duct by syringe, or the introduction of an abnormal bile (infected, concentrated, mucin-free) under a pressure approximating the physiological maximum.

Due prominence must be given to these facts as constituting a basis of rational treatment.

GENERAL DATA.

Of the 49 cases under review, 20 occurred in males and 29 in females. The ages of the patients ranged from 15 (male, died) to 72 (male, recovered). Gall stones were definitely noted in 30 (61%) and as definitely absent, at operation or autopsy, in eight others. In two cases the gall bladder was buried in such dense adhesions that the fact could not be determined, but the remaining histories are not informative upon this point. In one case a previous cholecystogram had shown a non-filling gall bladder. Duct stones were noted in six cases (hepatic duct one, common duct five). In three of these a stone was impacted at the ampulla. In yet another case a stone was recovered from the first post-operative motion. Three patients gave a history of alcohol taken to excess. One instance of duodenal diverticulum, apparently quite incidental, was met. On a number of occasions the gall bladder was noted to be distended with thick dark bile, with or without gall stones.

An observation of great interest and importance is that in several instances the common duct was found considerably distended, even to the degree of permitting little finger exploration, and yet no stone could be demonstrated therein. The bile which escaped on incision of the duct frequently carried with it mucinous flakes or flocculi.

TYPES OF PANCREATIC LESION.

1. *Acute Pancreatic Œdema.*—There were nine cases (18·4%). The earliest and, in very mild cases, the sole gross manifestation of pancreatic involvement is the appearance of an œdema, manifest to the eye as a glassy swelling of the subperitoneal tissues over the pancreas and in its immediate neighbourhood—the neck of the gall bladder, the free edge of the gastro-hepatic omentum, and the origin of the transverse mesocolon. Enlargement and increased firmness of the pancreas are readily detected by direct palpation. The œdema may be distinctly bile stained and a peritoneal effusion, which sometimes accompanies it, may be similarly tinged.

No macroscopic evidence of pancreatic necrosis or hæmorrhage is seen. If this evidence be present the condition is obviously removed from the category of simple pancreatic œdema, but a connecting link is provided by the fact that minor evidences of fat necrosis are not uncommonly found in the immediate vicinity. It is difficult to draw a sharp line of demarcation between such a condition, œdema with minor fat necrosis, and the milder cases of acute pancreatitis or pancreatic necrosis, and the inclusion of this œdema type of pancreatic lesion will undoubtedly react favourably upon the general recovery rate when examples of it are grouped statistically with the more serious fully established disease. An acute œdema of this nature was first described by Zoepffel,[6] who reported four cases, two with accompanying fat necrosis, with recovery in all. Sections taken at the time of operation showed an absence of hæmorrhage or necrosis of pancreatic cells. The condition is entirely analogous to that produced experimentally by Archibald following the introduction of clean bile from the gall bladder of a cat into its pancreatic duct, and he has suggested[7] that such a lesion probably furnishes the explanation of many attacks of upper abdominal pain of doubtful origin.

During the latter part of the period under review we have met with nine instances of this type of lesion and have been able not only to recognize it before operation but to separate it with comparative certainty from the hæmorrhagic type. Two of these cases ended fatally from causes not directly connected with the pancreatic lesion—one from uræmia, the other from intestinal obstruction. In the latter case, autopsy twenty-nine days after operation showed the pancreas to be normal both macroscopically and microscopically. Gall stones were present in all nine cases. In not a few instances the subject of a clear-cut pancreatitis gives a history of previous attacks strongly suggestive of acute œdema. We believe that recognition of this type of pancreatic lesion as a definite clinical entity is of some importance. Not only does it furnish, in our experience, definite evidence of underlying biliary disease, but it should be regarded as a warning that graver disease of the pancreas may arise.

2. *Acute Hæmorrhagic Pancreatitis (Acute Cellular Pancreatic Necrosis).*—There were in the series thirty-four cases (69·4%) of acute hæmorrhagic pancreatitis (acute cellular pancreatic necrosis).

The striking features of the fully developed picture are the occurrence of fat necrosis and what has been described as a "hæmorrhagic tendency". The fat necrosis, a lipase saponification of those fatty tissues to which the ferment has gained access, is almost invariably confined to the abdomen and is usually more or less juxta-pancreatic, although at times the change may be almost general throughout the abdomen. In two cases in this series necrosis has been observed elsewhere. In both these, of fulminant type and with the patients moribund on admission, numerous plaques were found scattered beneath the left pleura, and in one the subpericardial fat showed slight changes of this nature. The

distribution of the necrotic areas suggests that dissemination of the enzyme in effective amounts is by direct or lymphatic spread rather than by the blood stream. Much of the great omentum in another patient, the usual stout woman of middle age, the subject of a mild attack a fortnight earlier, was found necrotic and forming a mass larger than a coconut extending from the costal margin to below the umbilicus. It is unusual to find such massive involvement.

"Hæmorrhagic tendency" is an unfortunate term, inasmuch as it has been used to include certain hæmorrhages, entirely incidental, and due to an associated jaundice of some duration. Of this description are the cutaneous extravasations and tendency to post-operative oozing that may rarely occur. Only one instance of pre-existent deep and prolonged jaundice has been met in the cases under review. The hæmorrhage proper to the condition may either involve the pancreas alone or extend retroperitoneally beyond the immediate confines of the gland, not uncommonly into the attachments of the mesenteries and occasionally to surround the kidney and ureter. The extravasation may be so extensive as to obscure the pancreatic outlines or even to burst through into the lesser sac. With the more usual lesser degrees of hæmorrhage a marbled effect is produced, patchy extravasation contrasting with the paler gland tissue, or areas of fat necrosis standing out against a blood-stained background.

Such manifestations may not be uniform throughout the gland, the head or tail alone at times being found involved, a point of some importance; and the impression has been gained that such hæmorrhages are mainly superficial. At the same time, in many cases a more or less sanguineous peritoneal effusion occurs, either generally throughout the peritoneal cavity or confined to the lesser sac.

It is important to recognize that a fatal pancreatic lesion may be associated with a peritoneal effusion which is no more than bile stained. In one instance the effusion was both bile and blood stained. Other hæmorrhages directly associated with the disease are the hæmatemeses sometimes seen at the onset or during the course of the illness (three cases), and the secondary hæmorrhages which may occur during the separation of fatty or other sloughs (three cases).

3. *Acute Gross Pancreatic Necrosis.*

4. *Suppurative Pancreatitis.*

There were three cases of acute gross pancreatic necrosis and three cases of suppurative pancreatitis (12·2% for both types combined).

These types may be given joint consideration. In every instance the condition was discovery at autopsy, and in only one instance had any operation been performed. In the other five either an error in diagnosis (for example, coronary thrombosis) was made or the general condition of the patient forbade surgical interference.

A study of these cases is instructive. The one laparotomy was made three days after the onset of illness, a swollen pancreas and a clear peri-

toneal exudate being found. The only treatment consisted in the insertion of a tube drain to the pancreas (site unspecified). No gall stones were present and no biliary decompression was carried out. Death followed two days later and autopsy then showed that the lesion had advanced from a condition of pancreatic œdema to one of "inflammation and necrosis of the head of the pancreas". Two other massive necroses were revealed at autopsy, the durations of illness being 21 and 26 days respectively. In the records of these examinations nothing suggests the finding of any infective process in the pancreas. The *post mortem* findings in the three cases of suppurative pancreatitis were as follows: (i) "multiple suppurating areas in the pancreas", after an illness given as of "some weeks"; (ii) "a general peritonitis", after five weeks; and (iii) "a suppurative pancreatitis, lesser sac full of pus", twelve days after the onset of the disease.

It would appear from the above that gross necrosis comes into the picture of the disease only after a considerable interval, and evident infection usually still later in the neglected case.

That no instance of a definite suppurative process was found in the pancreas before the lapse of twelve days (although operation in several other cases was not performed until the fifth, sixth or seventh day of illness) suggests that the infective condition rather than being one of primary association or directly causative, is probably due to a secondary and incidental invasion of necrotic fat or other devitalized tissue. In confirmation of this suggestion is the fact that in no case in which operation included a satisfactory biliary decompression, but in which death resulted later, has any more advanced lesion of the pancreas itself been demonstrated at autopsy than was noted at operation. On the contrary, remarkable powers of repair seem peculiar to the pancreas. Thus in a case of subacute pancreatitis (not included in the series) the tail was still firm and flecked with hæmorrhage and fat necrosis from an attack three weeks earlier, yet the section of gland tissue removed at operation (cholecystectomy and choledochotomy) showed a completely normal alveolar structure. Similarly, the pancreas has been found both at autopsy five weeks after operation and on subsequent microscopical study merely to carry traces of old superficial hæmorrhage and fat necrosis, although the tail was actually passing through an undetected collection of pus and necrotic fat below the spleen. Five weeks earlier "hæmorrhage and necrotic spoiling" had been observed. Reference has already been made to a patient with pancreatic œdema dying twenty-nine days after operation from obstruction of the small bowel. Here, too, microscopical examination of sections removed at autopsy revealed no abnormality.

It is significant that no further instance of gross pancreatic sloughing or suppuration has been unexpectedly revealed at operation or

autopsy since the routine estimation of urinary diastase in all acute upper abdominal affections has been carried out.[1]

PROGRESS OF THE DISEASE.

Irrespective of the precise agent responsible for the autolysis, intensely toxic bodies of protein origin and possibly of histamine type are formed. These products are in large part at any rate responsible for the extreme collapse seen in certain cases of the disease. Such patients may never rally sufficiently for operation to become practicable, or may die within twelve to twenty-four hours if one be performed. In those less fulminant cases in which the patients weather the initial storm and yet fail to convalesce promptly after operation, a slow secondary infection of necrotic tissues may lead to the insidious formation of abscesses of a curious type. The content is a greyish glutinous mixture of pus and broken-down fat containing fragments of necrotic (saponified) fat of various sizes. These sloughs, often termed pancreatic, may continue to be discharged for many months, until either recovery finally comes about or death from chronic sepsis ends the scene. Three patients in the present series succumbed after illnesses of three, five and six months. Such abscesses appear to have a special predilection for the region of the tail of the pancreas, but may be found anywhere in relation to necrotic fat. Commonly enough, the patient is completely afebrile, though obviously not progressing. The incision shows no proper signs of repair and tends to open. A slimy and purulent biliary discharge appears to be responsible for this, even though no pancreatic ferments can be demonstrated therein. Vomiting, which begins days or weeks after the operation and which yields to no treatment, is most suggestive. Tenderness or the formation of a vague mass in the left hypochondrium or elsewhere will leave no room for doubt that further operation for drainage is imperative.

SYMPTOMATOLOGY.

Prodromata.—In most histories a story of previous attacks of pain, usually much less severe than the present, is elicited. Such are generally ascribed to gall stones, and this explanation is probably correct in the

[1] Schmieden and Sebening give the percentage incidence of the types of the disease and the corresponding mortality rates as:

Percentage Incidence.	Type of Disease.	Cured.	Died.
9·2	Œdema without fat necrosis.	76·3	23·7
35·4	Œdema with fat necrosis.	62·5	37·5
25·1	Hæmorrhagic infarcts.	36·1	63·9
19·3	Necrosis with areas of softening.	32·0	68·0
3·4	Sequestration.	43·1	56·9
7·6	Abscess formation.	39·9	60·1
		48·8	51·2

majority, for often there has been an accompanying jaundice. In others, again, it seems likely, from the description of the pain and its association with some collapse, that mild attacks of pancreatitis or pancreatic œdema have been experienced. In only four cases of the present series is it definitely stated that no previous attacks of pain or upper abdominal distress had occurred at any time. Schmieden and Sebening stated that in their experience cases without prodromata are the most fatal. Of our four patients, two recovered and two died. A previous operation upon the biliary tract had actually been performed in four instances; reference to these will be made later.

That attacks of apparently fairly definite biliary colic have occurred in the past is certainly of great value once a suspicion of pancreatitis has been aroused, but without this it may seriously prejudice the chances of recovery in that merely palliative treatment may be instituted.

Pain.—This symptom is one which, more than any other, is worthy of the closest attention as furnishing most valuable guidance. If failure of relief by morphia can be taken as an index of severity, it is doubtful if a greater agony exists. Dick[8] and other writers have drawn attention to the inadequacy of the drug in ordinary dosage, and I have known an experienced ward sister make a confident and correct diagnosis on this feature alone. In but a single instance of this series was marked pain absent, epigastric discomfort and vomiting alone initiating the attack. In the majority of cases the onset is sudden, though flatulence of unusual severity may have preceded it. The pain may be continuously severe or attacks of increasing severity may recur, culminating in a final agony of colic. Or the attacks, each lasting for a number of hours, may be separated by distinct remissions or intermissions apart from the use of morphia. The pain is usually referred primarily to the epigastrium, one or other hypochondrium, or the upper abdomen in general. In one case alone was the pain referred to the lower abdomen, and in this all tenderness was upper abdominal. In another the pain was umbilical at onset, soon moving to the right hypochondrium. The pain is at times exaggerated on inspiration with complaint of "catching the breath". Especially significant is an epigastric pain radiating to the left. Even more characteristic, and particularly important in diagnosis, is a dorsal radiation of the pain, to "the back" generally, to the lumbar region or to the left shoulder blade. It is impossible to place too much emphasis upon this feature (dorsal radiation) which, although definitely noted as present in only ten histories, was actually far more frequent. The description given is of a pain, not in the back, but going through to the back, loin or shoulder as the case may be.

The pain has been variously ascribed to pressure upon and involvement of the cœliac plexus, hæmorrhage into the mesocolon, the shedding of enzymes into the peritoneal cavity and distension of the gland capsule. Since the pancreas possesses no capsule and the overlying peritoneum can often be demonstrated by the finger to be movable upon the hæmorrhagic gland beneath, the last suggestion is untenable. Any history of

remissions is difficult to reconcile with the theories of enzyme peritonism or hæmorrhage into the mesocolon (which is by no means a constant finding). The history of the following case is given in support of the view which I have come to hold, that the pain is entirely due (apart from any later infective complications) to raised intraductal pressure and comparable therefore in origin (and severity) with biliary and renal colics.

K.L., aged sixty-one years, was admitted on February 18, 1931, two days after the onset of prostrating upper abdominal pain, with vomiting which continued up to the morning of admission. The pain had also been continuously present in spite of the use of morphia, which relieved it only partly, although half a grain dosage had been used. It remained as a dull ache, with colicky exacerbations on the second day, the pain passing through to the back, but never to either shoulder. Constipation had been complete since the onset. A history was given of an attack of colicky upper abdominal pain six months ago, lasting for an hour, without vomiting. There had been a similar attack six weeks ago, lasting for three hours. The temperature was found to be 38° C. (100·4° F.), the pulse rate 88, respiratory rate 24.

On examination there was no jaundice, the tongue was clean, dry and red. The pulse was of good volume and tension. The abdomen was full or slightly distended, but with no rigidity. Tenderness was elicited on deep pressure in the mid-epigastrium, beneath the left costal margin and in the left loin. No mass was to be felt. The Loewe mydriasis test gave a doubtfully positive reaction after forty minutes. The urinary diastase was 2,000 units. The abdomen was opened under ethylene and oxygen anæsthesia, a free brownish-red fluid being liberated and fat necrosis of the extraperitoneal fat, the omentum and around the lesser sac, particularly the foramen of Winslow, being revealed. The pancreas was exposed through the lesser omentum and showed a small amount of hæmorrhage and "necrotic spoiling". It was definitely firm and enlarged. The gall bladder was thickened and tense. Bile was taken by needling from both gall bladder and common duct for culture. The thick dark gall bladder bile later showed a growth of *Bacillus alkaligenes*; bile from the common duct was sterile. The gall bladder containing one large stone was removed. The common duct was opened, shown to be patent, and then drained both up and down by tubes. No attack of any sort by incision or drainage was made upon the pancreas. Oozing from the gall bladder bed was checked by cigarette gauze drain. The usual rapid recovery of consciousness after ethylene anæsthesia brought with it the realization that all pain, except that resulting from the incision, had completely disappeared.

A similar observation has been made and recorded by us on at least three occasions. In no case was an acute inflammation of the gall bladder present, removal of which might be held responsible for the relief. We believe that these observations are entitled to full consideration as having an intimate bearing upon both causation and treatment.

Tenderness.—Like pain, tenderness is always present unless the patient has been heavily morphinized. In general, it may be described as upper abdominal, and is usually noted as maximal in the epigastrium. Rarely the tenderness is more pronounced in the right hypochondrium. In the latter cases an associated gross cholecystic disease probably exists or the gall bladder is greatly distended. Most significant, however, is a tenderness maximal in the left hypochondrium, and also to be elicited by pressure in the left flank or loin. While tenderness is frequently present in both hypochondria, it is usually more pronounced in the left, "over the

situation of the pancreas", in the left flank or loin. It is to be noted that on account of marked right hypochondriac tenderness the most usual error in provisional diagnosis is acute cholecystitis. When tenderness is confined to the epigastrium and left hypochondrium alone, it is specially significant.

Vomiting.—Vomiting had occurred in variable degree in all cases save one. The patient in this case, a man of fifty-three years, had not even experienced nausea. He died three days after operation. A small hæmatemesis has been very occasionally noted.

Other Signs and Symptoms.—With the exception of the triad of pain, vomiting and tenderness, all other signs and symptoms are extraordinarily protean. Thus rigidity, which exceptionally may be so absolute as to simulate that of a perforation, in others is completely lacking, contrasting strangely with an exquisite tenderness. Perhaps more commonly than not, rigidity is present in some degree about the upper part of the abdomen on one or both sides. Marked rigidity does not necessarily signify an unusually grave lesion. It is sometimes present in the condition of pancreatic œdema, whilst in a fatal case of the hæmorrhagic type, associated with collapse, the abdomen may be perfectly flaccid.

Despite statements to the contrary, collapse is by no means constantly seen even at the outset. It is mentioned as being present, or having been present, in nine cases, in six of which the patient came to operation with but a single recovery. Thus it is of grave prognostic import.

It is noteworthy that an apparently good general condition may pass very easily and rather unexpectedly into a condition of collapse after operation, and thus it is by no means always easy to assess the operative risk. The pulse rate is frequently below 90, even in a serious case, and in two fatal instances the rates charted on admission were 66 and 72 respectively. The temperature at this time was perhaps most commonly in the neighbourhood of 37·8° C. (100° F.), but may be normal or subnormal. Constipation to the extent of failure to pass flatus following repeated enemata has been noted on several occasions. In such cases a diagnosis of intestinal obstruction has usually been made, a diagnosis in which the patient from his own sensations has heartily concurred, and at operation the transverse colon has been found distended. It seems that the pseudo-ileus of this portion of the bowel is associated with hæmorrhage spreading into the transverse mesocolon. Distension, however, is rarely at all general. The swollen pancreas is seldom palpable through the abdominal wall, although a suspicion may be aroused. Generally it is obscured either by a thick layer of subcutaneous fat or the guarding of the abdominal muscles. Jaundice, usually slight in degree, was present in five cases at the time of operation; in only one was it deep. Glycosuria was noted in three cases on the patient's admission. The tongue was frequently noted to be dry, or dry and furred, a condition possibly dependent upon the liberal use of morphine.

Signs and Symptoms Peculiar to the Disease.—Cyanosis. The general slight cyanotic tint, most obvious in the face, which is said to be peculiar to the disease, was noted on nine occasions, once by the patient herself. Six of these patients succumbed. That it is due, as is claimed by some, to any special toxæmia is very doubtful, for it is found in other upper abdominal lesions (perforation of gall bladder *et cetera*) and disappears with the relief of pain after operation. It is probably due to an anoxæmia from excessive diaphragmatic and rectus spasm. Localized lower abdominal cyanosis was noted on admission in one fulminating and fatal case.

The significance and value of those tests having special reference to pancreatic function will be fully dealt with by the following speaker. (It may be mentioned that Loewe's sign has been found entirely unreliable, but that the estimation of the urinary diastase has not failed to confirm or refute a clinical diagnosis of acute pancreatic disease.)

DIAGNOSIS.

The outstanding feature of all acute abdominal affections is pain, usually associated with tenderness and vomiting, and to this rule acute pancreatitis is no exception. In the differential diagnosis of the various emergencies it is by consideration of the location of such pain and tenderness, taken with the previous history and a study of associated signs and symptoms—vomiting, rigidity, collapse, constipation, jaundice, distension or the presence of some swelling or peritoneal effusion—that a conclusion is reached. The pictures presented by the varied lesions met are all made up by the foregoing in different combinations. As a rule, careful examination and due appraisement of the features of the case under consideration will lead to a correct pre-operative diagnosis. We submit that this statement is fully applicable to the disease in question despite the obscurity with which it is held to be surrounded.

Unless, however, one is prepared to discuss deliberately the possibility of this disease in the investigation of every acute upper abdominal affection, it is probable that cases of acute pancreatitis will continue to come to operation, sometimes at an unfortunately late stage, under some other diagnosis. "Unless a surgeon has seen previously two or three cases of acute pancreatitis or unless he keeps the condition constantly in mind, it is seldom that a correct diagnosis is made before opening the abdomen" (Deaver). "Even today we do not give enough attention to Koerte's old warning to consider acute pancreatic necrosis in all cases developing acute abdominal diseases, especially when biliary colic has been evidenced" (Schmieden and Sebening).

The position may be summed up in this way. The picture often enough appears to be an anomalous and contradictory one, but these very features should arose suspicion. If the possibility of the disease be borne in mind, and if the type and situation of the pain and tenderness be carefully elicited, very few cases will be missed. If the Wohlgemuth test for urinary diastase be carried out in all acute lesions of the upper part of the abdomen, none will be missed.

The hernial sac, be it inguinal, incisional or umbilical, which is found distended with a blood-stained effusion following an attack of abdominal pain and vomiting, is most likely to continue to be a source of error.

A provisional diagnosis of acute pancreatitis (qualified, in the pre-Wohlgemuth period, by a question mark, or with an alternative diagnosis attached) was made in 21 of the 45 hospital cases. On other history sheets, in that same period, appear such varied diagnoses as acute cholecystitis (8), perforated ulcer (4), intestinal obstruction (3), acute appendicitis, internal hæmorrhage, diaphragmatic pleurisy and perforation of the gall bladder (one of my private cases).

TREATMENT.

Any discussion of treatment must be prefaced by a further reference to the influence of chronic biliary disease in the genesis of pancreatitis. It is impossible to avoid the conclusion that timely treatment of the former would undoubtedly have saved many of our patients. In one instance the attack actually occurred between the finding of a non-filling gall bladder by cholecystography and the admission of the patient for operation. In four other cases a previous operation upon the biliary tract had been performed. In none of these had operation been complete and satisfactory, for in two the common duct still harboured a stone, in another a cholecystectomy had been found impossible, and in the fourth a calculous gall bladder had been removed, but the ducts had not been explored.

Furthermore, many disasters have been due to the wide acceptance of the teaching, from which I dissociate myself, that operation for gall stones or cholecystitis should be undertaken only when acute symptoms have subsided. Only too often has a pancreatitis been regarded as acute cholecystitis or severe biliary colic, and precious hours and days have been lost in palliative and expectant treatment.

The uncertainty which exists as to the ætiology of the disease has inevitably brought in its train confused, ill-defined and illogical methods of treatment. The very term "pancreatitis" is not blameless in this respect.

John B. Deaver[5] himself has written:

> The sequence of events is cholecystitis leading to pancreatic lymphangitis . . . . therefore, as elsewhere in the body systems, treatment of the primary focus of infection is the rational step to pursue.
>
> Instant drainage of the pancreas is the procedure indicated. . . . Any further surgery depends on circumstances. . . . The condition of the patient permitting, attention to the bile passages and the gall bladder is not out of place. . . . Pancreatostomy is beneficial if for no other reason than the relief of tension it affords.

He advocates free incision of the overlying peritoneum and a few blunt punctures into the pancreas.

I have already expressed the belief that the lymphatic infective causation is difficult of acceptance and that the canalicular theory is

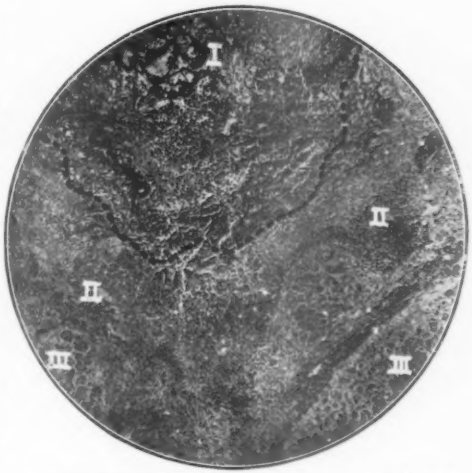
almost entirely satisfactory both experimentally and clinically. I have called attention to certain clinical observations in its support:

(1) It is difficult to reconcile the sudden onset seen in many cases, or any history of remissions and intermissions, with an inflammatory process.

(2) The not very rare localization of the disease to the tail, or exemption of the head, speaks against any lymphatic spread from the gall bladder.

(3) The relief of pain observed to follow decompression of the duct system.

(4) The dilatation of gall bladder and common duct frequently seen, both at operation and autopsy, even in the absence of common duct stone. Colp[9] has made the same observation in seven of his 51 reported cases.



Photomicrograph of pancreas (× 20). I = deeply bile stained necrotic pancreatic tissue, II = hæmorrhage, III = living pancreatic lobules.

(5) Direct evidence of biliary extravasation has been observed. (*a*) Bile staining of peritoneal effusion, or presence of a similar œdema (six cases). (*b*) Bile staining of the pancreatic duct has been noted *post mortem*, and in one case gross staining of the pancreatic parenchyma (see figure). (*c*) Biliary discharge was noted in one case of protracted illness, with much sloughing from the region of the pancreas, at a time when the cholecystostomy was demonstrated by further operation to be healed. The ducts in this case had not been explored. (A somewhat similar observation has been made by Timbrell Fisher.[10] In his case operation had been strictly confined to the pancreas.)

In view of the foregoing it is difficult to avoid the conclusion that retrojection of an abnormal bile must be regarded as causative. That being so, the primary aim of surgical treatment becomes immediately evident. It lies in biliary decompression, which, by preventing further regurgitation, will limit pancreatic damage.

Archibald writes:[4]

I think it may be concluded that the surgical treatment of pancreatitis must chiefly lie in a prevention of any further retrojection of bile into the pancreas.

The damage already done to the organ will often be cured by the processes of nature, absorption, replacement, or even sequestration. On the other hand, if gangrene, abscess or total slough has occurred, it may require a direct attack upon the pancreas itself. But to prevent recurrence or extension the cardinal point would seem to lie in the prevention of any heightened pressure in the biliary system and, secondly, in the restoration of an altered bile to its normal chemical condition, or of an infected bile to sterile bile. This can only be done by a drain in the gall bladder or in the common duct.

Hæmorrhagic effusion has been found within six and half hours of the onset of the illness, so that probably long before the abdomen has been opened much damage, often irreparable, has been done and further injury is in progress. At present surgery can do little or nothing to

TABLE II.

All Cases.

Operation.	Number of Patients Recovered.	Number of Deaths.	Remarks.
1. Drainage of pancreatic region ..	3	6	
2. Drainage of pancreatic region and cholecystostomy	3	6	
3. Cholecystostomy	3	1	
4. Cholecystectomy and choledochostomy	9	4	
5. Cholecystostomy, choledochostomy and drainage of pancreatic region ..	1	1	The recovery followed trans-duodenal choledochotomy.
6. Choledochotomy, choledocho-duodenostomy	1	0	
7. Laparotomy only	1	1	(*a*) Gall bladder and ducts inaccessible. (*b*) State of patient.
8. No operation	1	8	The recovery followed refusal of operation. Of the remainder, five were unfit for operation and the condition of three was unsuspected during life.
Totals	22	27	A general recovery rate of 44·9%. Operative, 52·5%.

avert the consequences that follow the free shedding of activated ferments into the areolar tissues around the pancreas. Ferments shed into the peritoneal cavity can be evacuated, but not so those diffused along tissue planes and into tissue spaces. Incision through the peritoneal covering will succeed in liberating a mere fraction of the effusion present in the interstices there, and may be definitely harmful in addition. Free oozing follows, particularly if the pancreas itself be unwisely included in the attack, and gauze tamponade becomes necessary. The necessary firm packing carries with it the risk of pressure necrosis and establishes at the same time a track from the skin to devitalized tissues in the depths.

Only in those cases in which gangrene, abscess or total slough has occurred or seems inevitable, is direct attack upon the pancreas by peritoneal incision advisable. In these the illness will almost always have been of some duration.

I believe that at operation our aim should be: (i) to free both greater and lesser sacs of any effusion, particularly when blood stained: (ii) to bring about a satisfactory biliary (and thus pancreatic) decompression, usually by opening, exploring and draining the common duct: (iii) to remove the gall bladder, unless the state of the patient makes this prolongation of the operation unwise on general surgical grounds.

The first two indications are absolute. Many recoveries, including Halstead's classical case in 1901, the second on record, have followed simple removal of the effusion. This is always possible and may be brought about by the use of the evacuator or the free use of gauze

TABLE III.

Personal Cases.

Operation.	Number of Patients Recovered.	Number of Deaths.	Remarks.
1. Drainage of pancreatic region ..	1	0	
2. Drainage of pancreatic region and cholecystostomy	2	4	(*a*) 24 hours. (*b*) 24 hours. (*c*) 5 months. (*d*) 6 months.
3. Cholecystostomy	1	0	No gall stones; common duct inaccessible.
4. Cholecystectomy and choledochostomy	7	3	(*a*) 5 weeks. (*b*) On table. (*c*) 29 days, from intestinal obstruction.
5. Cholecystostomy, choledochotomy and drainage of pancreatic region ..	1	0	Transduodenal removal of impacted stone.
6. Choledochotomy, choledocho-duodenostomy	1	0	Stone believed to have slipped back into dilated pancreatic duct.
7. No operation	0	1	Moribund. Stone found impacted at ampulla at autopsy.
Totals	13	8	38·1% mortality.

sponges. When the exudate is large and general, a suprapubic stab drain may be advisable. This was done twice in the series. When previous operation upon the gall bladder has been performed, gross adhesions may necessitate a second left sided incision to give access to the lesser sac.

The finding and opening of the common duct are usually possible, but may be very difficult on account of old adhesions or huge swelling of the pancreatic head. For these reasons the attempt may have to be abandoned and the far less satisfactory gall bladder drainage instituted. It has been quite evident, from our experience both at the operation table and at autopsy, that cholecystostomy is the less satisfactory procedure, even when bile drainage appears to have been established. A third method of biliary and pancreatic decompression, obviating incision of the supraduodenal portion of the duct, is by transduodenal incision

of the ampulla. This method, which was adopted with success in one of my cases, is practicable only when a palpable stone is lodged there.

The third indication, cholecystectomy, is relative. It is conditioned by the operative risk presented by the patient at that stage. It is less concerned with the immediate saving of life than with the future welfare of the patient, particularly as regards immunity from further attacks. The potential source of a continued biliary infection or stone formation should be removed, if not at the time, at some later date. The extraordinary powers of repair inherent in the pancreas, of which I have given illustrations, are no doubt bound up with its huge arterial supply which reaches it from every adjacent source. Primary reliance should be placed upon these powers for restoration of the gland.

Only in the event of a finding of gross necrosis or an established frank suppurative process is tube drainage of the pancreas advocated. Treatment then follows general surgical principles and the posterior route to the pancreatic tail and lesser sac has obvious advantages.

Many patients with acute pancreatic necrosis will continue inevitably to perish, but earlier diagnosis, followed by suitable operative treatment, will, I am convinced, lead to a diminution of the present ghastly mortality.

To my colleagues on the surgical staff I wish to express my sincere thanks, not only for access to records of their cases, but for their kindly tolerance in permitting me to appropriate a number of patients with this most interesting disease from their beds to my own care.

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URINARY DIASTASE IN ACUTE PANCREATITIS.[1]

By M. Mushin.

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During a period of four years ending December, 1931, forty-five patients with acute pancreatitis were admitted to the Alfred Hospital. In the first two years the diagnosis rested entirely on the clinical findings. Because of the number of incorrect pre-operative diagnoses an attempt was made to establish a relatively simple and certain method of confirming the presence of pancreatitis. Accordingly, the last twenty-six patients were submitted to quantitative tests for urinary diastase, and from the results obtained in this and a representative control series, it appeared that this test possessed the element of certainty desired.

The usual investigations available for the detection of pancreatic inefficiency are those concerned with (i) mydriasis, (ii) changes in the fæces, (iii) changes in the urine.

The results of most of the investigations recommended are notoriously inconstant, and many quite unfit for practical application to the condition of acute pancreatitis by virtue of the complicated technique and length of time involved in their execution. This point is emphasized by the fact that in the majority of cases which form the subject of this discussion an immediate operation was pending. For these reasons, investigational methods giving constant results in a minimum length of time were preferred.

In the present series no examination of fæces was made owing to the lengthy and elaborate technique involved. The simpler urinary tests, such as those for the presence of glucose, diacetic acid, acetone and bile, were performed in a routine manner and yielded variable results. Thus bile was very frequently present due to the associated cholelithiasis, or the impaction of a gall stone in the ampulla of Vater. In the majority of cases there was no glycosuria or ketonuria.

Loewe's Mydriatic Test.

The technique of Loewe's mydriatic test consists in the instillation of two drops of adrenaline hydrochloride (one in 1,000) into one eye (having first noted the equality of the pupils) and repeating it in five minutes. Thirty minutes after the second instillation any appearance of dilatation of the pupil of this eye is noted. A dilated and usually fixed pupil denotes a positive result.

[1] Read at the annual meeting of the Royal Australasian College of Surgeons, February, 1932.

Of the 32 patients in the present series tested in this manner, only four gave positive results, approximately 13%. On the other hand, 150 patients suffering from miscellaneous medical and surgical conditions in the wards were submitted to this test with a positive result in ten instances, approximately 7%.

It is thus seen that Loewe's mydriatic test has little, if anything, to commend it as a means of diagnosis of acute pancreatitis, despite what has been stated so often by writers on this subject.

URINARY DIASTASE.

The technique[1] of the urinary diastase test as performed in the present series consisted in adding one cubic centimetre of normal saline solution to each of five or more numbered test tubes. To the first was then added one cubic centimetre of urine. One cubic centimetre of this diluted specimen was then added to the second test tube and so on down the series. This resulted in urine dilutions of $\frac{1}{2}$, $\frac{1}{4}$, $\frac{1}{8}$, $\frac{1}{16}$, $\frac{1}{32}$ *et cetera*. To each test tube was then added two cubic centimetres of 0·1% starch solution. All test tubes were then placed in an incubator at 37° C. for 30 minutes. At the end of this time cold water was added to each (to retard further digestion) followed by a few drops of tincture of iodine.

Owing to the successive dilutions of urine, the amount of diastase present in the consecutive test tubes for the digestion of starch suffered a rapid and proportional diminution.

Depending on the amount of diastase present, after thirty minutes' incubation certain test tubes would contain maltose (complete starch digestion), erythro- and achroo-dextrin (incomplete digestion) and starch (undigested owing to insufficient diastase, that is, too great a dilution of urine). On the addition of iodine these test tubes showed colours which were pale brown, reddish-brown, bluish-red or blue respectively. The end point was shown by a reddish-brown colour immediately preceding the test tube in which a bluish coloration was present.

Now a Wohlgemuth unit of diastase is given by the number of cubic centimetres of 0·1% starch solution digested by one cubic centi-

[1] It takes about 40 minutes (including the 30 minutes' incubation) to perform the urinary diastase test, and, as no elaborate apparatus is required, the technique and time spent in its performance are both within the reasonable limits allowed by the preliminary preparations for operation on an acute abdominal condition.

In the present series, suitably graduated burettes for the saline and starch solution, and a pipette for dilutions were used in each case. However, in the absence of these facilities, it was found that a "diagnostic" reading could be obtained in a much simpler manner as follows. A one in 25 dilution of urine was prepared by adding one cubic centimetre of urine to 24 cubic centimetres of saline solution, one cubic centimetre and five cubic centimetre or ten cubic centimetre graduated syringes being used. One cubic centimetre of this diluted urine was now added to a small glass tube fitted with a stopper, for example, a Wassermann tube, and two cubic centimetres of 0·1% starch solution added. The tube was then placed in a vest pocket and the time noted. In about five minutes a small quantity was extracted and tested with a drop of iodine. If a blue colour resulted, the tube was "incubated" for a further five minutes and again tested, and so on until a test revealed a brown or reddish-brown colour, but not blue. This represented the end point, and meant that if attained within 30 minutes there were at least $\frac{2}{1/25}$ = 50 units present. The test was thus often reduced by 15 or 20 minutes. Although a quantitative estimation was not made, a significant increase in urinary diastase was established.

metre of urine in thirty minutes at 37° C. Thus if the fifth test tube represented the end point, then the amount of diastase in one cubic centimetre of urine was: $\frac{2}{\frac{1}{32}} \frac{\text{(cubic centimetres of starch used)}}{\text{(dilution of urine)}} = 64$ units; if the tenth test tube, 2,048 units, and so on.

As a control the Wohlgemuth test was applied to 140 patients, including 115 with acute abdominal conditions and 25 with miscellaneous medical and surgical conditions unrelated to the biliary duct system. The former group included patients with acute cholecystitis, cholelithiasis and stone in the common bile duct, intestinal obstruction, acute appendicitis *et cetera*, the diagnosis in the majority being established by operation, and in the others by general progress or *post mortem* examination. In all, that is, medical and surgical conditions, the diastase content of the urine gave values between two and 32 units, the majority showing a value of 10 units.

Among this control series were three patients with pneumococcal peritonitis[1] and many others with conditions clinically resembling that of acute pancreatitis.

Two cases absolutely simulating acute pancreatitis, but which showed a normal urinary diastase, emphasized the value of this test. The first patient, who was cyanosed, was found at operation to have a perforated gangrenous gall bladder with general peritonitis and a large collection of turbid bile in the left hypochondrium. The second had ruptured an amœbic abscess of the liver with the subsequent development of general peritonitis and contamination of the lesser omental sac.

In the present series of 45 patients suffering from acute pancreatitis, only the last 26 were submitted to a urinary diastase test, as this test was finally adopted as a routine in suspected cases in the latter half of the four years' period of investigation only.

From Table I it will be seen that with the exception of Case I, all the proven cases of acute pancreatitis of whatever type showed a urinary diastase value between 50 and 4,100 units in this latter period. (Owing to a provisional misdiagnosis, the urinary diastase in Case I was not determined until ten days after admission, by which time it returned to normal, as is shown in Table IV.) Further, it will be noted that in 73% of cases readings of over 100 units and in 27% readings of under 100 units were obtained.

In the first half of the period of investigation, when urinary diastase tests were not performed in cases of doubtful upper abdominal crises

[1] R. J. McNeill Love, reviewing 51 cases of acute pancreatitis in *The Lancet*, December 18, 1926, makes the following statement: "The diastase index, normally 10 to 20 units, has obviously little diagnostic value in emergency surgery, although in one case in this series 100 units were present. Also other acute abdominal conditions may diminish the value of this test by raising the index; thus Barling records a case of pneumococcal peritonitis with a diastase index of 50 units." We have been unable to confirm this.

TABLE I.

Case Number.	Urinary Diastase.	Pre-operative Duration.	Operative Diagnosis.	Result	Remarks.
I	16	10 days	Acute pancreatitis, fat necrosis.	Cure.	
II	50	3 days.	Acute hæmorrhagic pancreatitis.	Died.	
III	64	? 6 days.	Acute hæmorrhagic pancreatitis.	Died.	
IV	64	3 days.	Acute hæmorrhagic pancreatitis.	Died.	
* V	64	7 hours.	Acute pancreatic œdema.	Cure.	
VI	66	5 days.	Acute pancreatic necrosis.	Cure.	
VII	66	2 days.	Acute pancreatic œdema.	Cure.	27% under 100 units.
VIII	128	16 hours.	Acute pancreatic œdema.	Cure.	
IX	160	2 days.	Acute pancreatitis, fat necrosis.	Cure.	
X	240	36 hours.	Acute pancreatic œdema.	Cure.	
* XI	256	11 hours.	Acute pancreatitis, fat necrosis.	Cure.	
XII	256	3 days.	Acute pancreatitis, fat necrosis.	Cure.	
XIII	300	2 days.	Acute pancreatitis.	Died.	
XIV	350	? 5 days, ? 1 day	Acute pancreatic œdema.	Cure.	
XV	360	? 5 days.	Acute pancreatitis.	Cure.	
XVI	400	7 hours.	Acute pancreatic œdema.	Cure.	
XVII	500	3 days.	Acute pancreatic œdema (uræmia).	Died.	
XVIII	512	2 days.	Acute hæmorrhagic pancreatitis	Died.	
XIX	700	2 days.	Acute pancreatic œdema.	Cure.	
XX	750	12 hours.	Acute hæmorrhagic pancreatitis.	Died.	
XXI	2,000	36 hours.	Acute pancreatic œdema.	Cure.	
XXII	2,000	24 hours.	Acute pancreatic œdema (intestinal obstruction).	Died.	
XXIII	2,000	2 days.	Acute pancreatitis, fat necrosis (pneumonia)	Died.	
XXIV	2,048	2 days.	Acute pancreatic fat necrosis.	Cure.	
XXV	4,100	10 hours.	Acute hæmorrhagic pancreatitis.	Died.	
XXVI	4,100	14 hours.	Acute pancreatitis, fat necrosis.	Cure.	73% over 100 units.

* Cases V and XI are referable to the same patient. In Case V the condition of the patient and operative difficulties rendered possible a cholecystostomy only. Case XI refers to his readmission with acute pancreatitis some two months later.

TABLE IIA.

Showing Urinary Diastase (Expressed in Units) of Dogs.

Animal.	Urinary Diastase, Pre-operative.	Urinary Diastase One Day after Operation.	Urinary Diastase, Two Days after Operation.	Diagnosis *Post Mortem*.
Dog I	4	150	64	Acute hæmorrhagic pancreatitis.
„ II	8	64	128	Acute hæmorrhagic pancreatitis.
„ III	2	128	128	Acute hæmorrhagic pancreatitis.
„ IV	4	128	32	Acute hæmorrhagic pancreatitis.
„ V	4	32	32	Acute hæmorrhagic pancreatitis.

TABLE IIB.

Showing Urinary Diastase (Expressed in Units) of Cats.

Animal.	Urinary Diastase, Pre-operative.	Urinary Diastase 30 Minutes after Operation.	Urinary Diastase 1 day after Operation.	Urinary Diastase 2 days after Operation.	Urinary Diastase 3 days after Operation.	Diagnosis *Post Mortem*.
Cat I ..	16	64	128	32	10	Acute pancreatic œdema, fat necrosis.
„ II ..	4	8	150	64	4	Acute pancreatic œdema, fat necrosis.

nor in cases of suspected pancreatitis, the pre-operative diagnosis of acute pancreatitis was present in only 30% of cases. So it happened that *post mortem* examinations from medical wards occasionally revealed an acute pancreatic condition of considerable duration, the provisional diagnosis having favoured such conditions as coronary thrombosis, angina *et cetera*. On the surgical side, this test, when employed, has always clinched the diagnosis in doubtful abdominal emergencies, and has been of particular value in acute gall bladder disease and in the toxic phase of intestinal obstruction and general peritonitis. It was our experience that in every abdominal crisis when the urinary diastase value was 50 or more units, operation confirmed the provisional diagnosis of acute pancreatitis; that is, correct in 100%.

Confirmation of the value of this test was sought in a small series of animal experiments in which pancreatitis was artificially induced in dogs and cats. Raised values, though to a less extent than in the clinical cases, were obtained in all cases. The following was the technique adopted:

> Under ether anæsthesia an upper mid-line incision was made and half a cubic centimetre of bile was aspirated from the gall bladder. An assistant meanwhile catheterized the bladder by means of a ureteric catheter. The bile was then injected into both the head and body of the pancreas and the wound was closed in the usual manner. Daily catheterization was performed until forty-eight hours after operation, when the animal was killed and a *post mortem* examination made. This revealed a small quantity of blood-stained fluid in the peritoneal cavity. Hæmorrhagic pancreatitis was present with widespread areas of fat necrosis.

These findings were present in all the experimental cases, although the lesions in cats were neither so gross nor so extensive as in dogs.

Tables IIA and IIB show the constant increase in urinary diastase following the experimental production of acute pancreatitis.

Returning to the present series, in Table III the urinary diastase values relative to the duration of the illness are shown. It is seen that up to forty-eight hours after the commencement of the pancreatitis, 88% showed values of over 100 units, whereas with a duration of three days or more only 44% reached that figure. Thus in the majority of cases the earlier the test was performed after the onset of the illness, the higher was the value obtained. In one experimental observation (Cat I, Table IIB) a definite increase was observed as early as thirty minutes after the commencement of the acute pancreatitis.

Early in the course of the series it was thought that a high diastase figure allowed of a more hopeful prognosis, but further study failed to substantiate this impression. (Compare Cases XXV and XXVI, Table I.) Little, if any, help relevant to the immediate prognosis is to be expected from the urinary diastase figure, which indicates functional impairment of the pancreas rather than the extent of its disease.

After the initial high figure the amount of urinary diastase usually fell rapidly to normal. This is shown in Table IV, where serial figures for consecutive days are given. As a rule, a normal reading resulted

TABLE III.

Case.	Duration.	Urinary Diastase. In Units.	Remarks.
I	7 hours.	64	Within 48 hours: 88% over 100 units. 53% over 500 units.
II	7 hours.	400	
III	10 hours.	4,100	
IV	11 hours.	256	
V	12 hours.	750	
VI	14 hours.	4,100	
VII	16 hours.	128	
VIII	24 hours.	2,000	
IX	36 hours.	240	
X	36 hours.	2,000	
XI	48 hours.	66	
XII	48 hours.	160	
XIII	48 hours.	300	
XIV	48 hours.	512	
XV	48 hours.	700	
XVI	48 hours.	2,000	
XVII	48 hours.	2,000	
XVIII	3 days.	50	Beyond 48 hours: 44% over 100 units. None over 500 units.
XIX	3 days.	64	
XX	3 days.	256	
XXI	3 days.	500	
XXII	5 days.	66	
XXIII	5 days.	350	
XXIV	5 days.	360	
XXV	6 days.	64	
XXVI	10 days.	16	

TABLE IV.

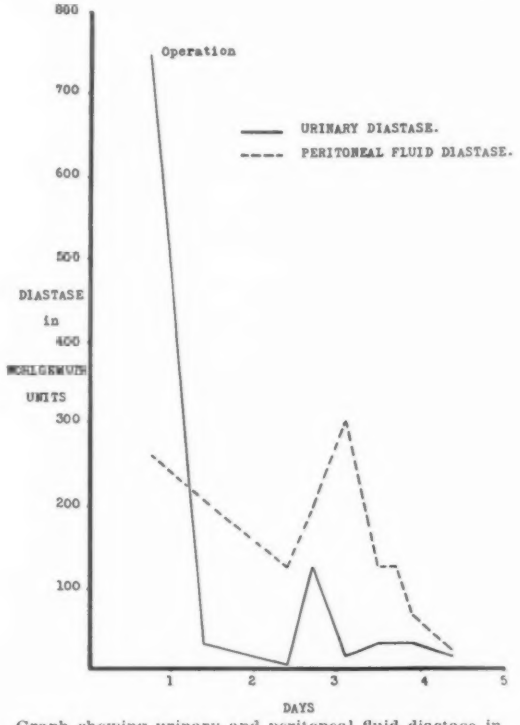
Urinary Diastase.

Case.	Before Operation.	First Day after Operation.	Second Day after Operation.	Third Day after Operation.	Fourth Day after Operation.	Remarks.
I	4,100	800	64	4	—	Decompression (cholecystectomy and choledochotomy).
II	2,000	60	16	8	—	Decompression (cholecystectomy and choledochotomy).
III	128	64	8	4	—	Decompression (cholecystectomy and choledochotomy).
IV	160	30	10	—	—	Decompression (cholecystectomy and choledochotomy).
V	350	2,000	220	50	16	Decompression (cholecystectomy and choledochotomy).
VI	700	60	8	8	—	Decompression (papilla incised).
VII	256	64	4	—	—	Decompression probably effective, though cholecystostomy alone performed.
VIII	750	128	32	32	16	Cholecystostomy (?) effective owing to incomplete removal of stones from Hartmann's pouch.
IX	360 (Tenth day before operation.)	124 (Ninth day before operation.)	50 (Third day before operation.)	40 (First day before operation.)	16 (First day after operation.)	Operation deferred on account of cardiac condition.

by about the third or fourth day following operation. Three interesting variations were observed:

1. The urinary diastase in Case V increased considerably on the day following operation and thereafter rapidly decreased in a normal manner. A gall stone was recovered from the first post-operative motion.

2. Case VIII (Table IV) was that of a patient operated on twelve hours after the onset of acute hæmorrhagic pancreatitis. Cholecystostomy was performed



Graph showing urinary and peritoneal fluid diastase in Case VIII (Table IV).

and another tube was inserted into the head of the pancreas. One day after operation the patient complained of upper abdominal colicky pain. The marked increase in urinary and peritoneal fluid diastase are shown in the accompanying graph. *Post mortem* examination eight days after operation showed that the gall bladder contained soft pigment sontes. It was thus doubtful whether the anticipated decompression of the biliary tract was effective.

3. Operation in Case IX (Table IV) was deferred for ten days owing to a mild degree of cardiac failure. The progressive decrease in the urinary diastase showed a definite "lag".

Thus it would seem from Table IV that some relationship exists between the degree of biliary decompression and the urinary diastatic index.

In a number of cases specimens of fluid from the peritoneal cavity were collected immediately the parietal peritoneum was incised in order to avoid contamination by blood. The diastase index was estimated by the technique outlined above. As shown in Table V, in the majority of instances the figures obtained were approximately half that of the urinary diastase.

TABLE V.

Case.	Urinary Diastase. In Units.	Peritoneal Fluid Diastase. In Units.
I	700	60
II	750	260
III	256	300
IV	4,100	2,048
V	4,100	1,024
VI	2,048	1,024
VII	512	256

CONCLUSIONS.

1. Operation or *post mortem* examination in 140 cases showing a normal urinary diastase[1] revealed conditions other than that of acute pancreatitis.

2. In a series of 26 cases each with a urinary diastase of 50 to 4,100, operation, or in one case *post mortem* examination, confirmed the provisional diagnosis of acute pancreatitis.

3. In general, the sooner the test was performed after the onset of the attack of acute pancreatitis, the higher the figure obtained.

4. Post-operative estimations showed a progressive and rapid diminution in the diastase index, except in those patients in whom effective biliary decompression was not apparently realized.

[1] Since the compilation of this paper, Mr. Quick has operated on a patient with acute pancreatitis in whom both urinary and peritoneal fluid diastase indices were normal.

OBSERVATIONS ON GLUCOSE TOLERANCE IN CASES OF RECOVERED PANCREATITIS.

By Ewen Downie.

[From the Baker Institute for Medical Research, Alfred Hospital, Melbourne.]

Despite the close relationship which exists between the pancreas and *diabetes mellitus*, the development of this disease as a sequel to acute pancreatitis is a rare event. Warfield[1] in 1927 reported four such cases, and could find reference in the literature to only seven others. Bernhard[2] in 1931 reported five cases in a series of fifty proved cases of pancreatitis. He states that there is a 10% risk of subsequent diabetes in the recovered cases. In two of his five cases, however, the evidence seems insufficient to assume the development of true *diabetes mellitus*.

At first sight it appears curious that a destructive and inflammatory lesion of the pancreas should not be associated more frequently with some evidence of impaired function of the islands of Langerhans. The accepted explanation lies in the fact that diabetes cannot be produced experimentally in animals by extirpation of the pancreas if one-sixth of the gland is left *in situ*. Warren[3] is no doubt correct in his statement that:

> Probably cases of diabetes after acute pancreatitis would be more frequent were it not that the lesions which destroy enough of the pancreas to produce insulin deficiency are generally sufficiently severe to cause death from the pancreatitis itself before there had been opportunity for the diabetes to demonstrate itself.

Certain observers have endeavoured to attribute the later development of diabetes to the site of the lesion in the pancreas. Thus Warfield states that no case of diabetes has followed a pancreatitis affecting the head of the organ. He seeks an explanation in the accepted anatomical fact that the islet tissue is found mostly in the tail of the gland. Another interesting speculation can be made from the work of de Takats,[4] who has shown that following ligation of the tail of the pancreas, an actual increase in the islet tissue occurs despite the fact that the other glandular elements undergo an atrophy. It is possible that the islet tissue is more resistant to the processes of inflammation which occur in acute pancreatitis than are the other parts of the organ.

During the course of investigations which have been carried out in the Alfred Hospital by Mr. Balcombe Quick and Dr. M. Mushin on certain aspects of acute pancreatitis, it occurred to the writer that an investigation of the glucose tolerance of the recovered patients might prove of some interest.

Eleven such patients have been submitted to a routine glucose tolerance test following the administration of fifty grammes of glucose by mouth. The estimations of the blood sugar were performed by Maclean's method in every case.

The findings in this series are shown in detail in the accompanying table. (The case numbers correspond to those in Table I in the paper by Dr. M. Mushin on urinary diastase. This gives brief details of the case histories.) The results of the investigations can be summarized thus: Of eleven patients examined, seven were females, four were males. In eight cases normal glucose tolerance curves were obtained. In three cases abnormal glucose tolerance curves were obtained.

The cases in which normal findings were obtained call for no comment and will not be considered further.

Of the three abnormal cases, one showed a temporary disturbance of tolerance which later returned to normal; one showed a temporary disturbance of tolerance which later returned to normal, but was associated with a lowered renal threshold to sugar; in one the patient developed a true *diabetes mellitus*.

These three cases will be considered in more detail.

CASE VI.—A female, aged forty-nine years, was operated upon in May, 1929. Her condition at the time of operation was so serious that a drainage of the pancreas was performed and no attempt was made to deal with a chronically infected gall bladder. Glycosuria was observed soon after the operation and a fasting blood sugar was found to be definitely raised. Six weeks after the operation a glucose tolerance test revealed a considerable departure from the normal. She was placed on a suitable diet and the glycosuria disappeared. On this *régime* she continued for one year. At the end of that time the fasting blood sugar was still definitely raised, and it appeared that her condition was that of a true *diabetes mellitus*. She was then operated upon a second time and a cholecystectomy was performed. Subsequently she continued with a dietary restriction for eighteen months, during which she remained free from any symptoms of diabetes. The urine during this period was repeatedly tested for sugar, but none was found at any time. A tolerance curve was then plotted and a normal result was obtained.

It is possible that the presence of an infected gall bladder was responsible for the impaired tolerance observed earlier in this illness. A similar example of this kind has been recorded elsewhere.[5]

CASE X.—A male, aged forty-eight years, was operated upon in June, 1931. Glycosuria had been observed in this case at intervals for several years before operation, but there were no symptoms of diabetes and no treatment had been instituted. Glycosuria was found on admission to hospital and four days after operation a tolerance curve showed a slight but definite departure from the normal. On his discharge he was recommended a dietary restriction which he did not follow because he felt so well. A tolerance curve seven months after the first one showed a normal result, but there was a definite glycosuria following the administration of glucose.

It is possible that this lowered renal threshold to sugar existed before operation and that, following the pancreatitis, there occurred a mild disturbance of tolerance which later subsided, leaving the condition of renal glycosuria as before.

TABLE OF PATIENTS SUBMITTED TO GLUCOSE TOLERANCE TEST.

Case Number.	Age in Years.	Sex.	Date of Operation.	Glucose Tolerance Test.							Remarks.
				Date.	Fasting Percentage.	½ hr. Percentage.	1 hr. Percentage.	1½ hrs. Percentage.	2 hrs. Percentage.	Glycosuria.	
VI	49	F.	May, 1929.	May, 1929.	0·26					+	12 days after drainage.
				June, 1929.	0·13	0·20	0·26	0·23	0·22	+	6 weeks after drainage.
			June, 1930.	June, 1930.	0·17					+	Before cholecystectomy.
				January, 1932.	0·09	0·15	0·17	0·14	0·10	0	Normal.
VII	66	F.	October, 1930.	January, 1932.	0·10	0·13	0·15	0·11	0·08	0	Normal.
X	48	M.	June, 1931.	June, 1931.	0·10	0·20	0·18	0·17	0·16	+	4 days after operation.
				January, 1932	0·10	0·12	0·16	0·13	0·09	+	Low renal threshold.
XII	33	F.	November 1931.	January, 1932.	0·08	0·11	0·17	0·14	0·10	0	Normal.
XIV	24	F.	December, 1930.	January, 1932.	0·09	0·14	0·11	0·10	0·10	0	Normal.
XVI	72	M.	April 1930.	January, 1932.	0·11	0·16	0·18	0·13	0·10	0	Normal.
XIX	72	F.	December, 1930.	January, 1932.	0·09	0·14	0·17	0·13	0·10	0	Normal.
XXI	52	M.	June, 1931.	January, 1932.	0·10	0·13	0·11	0·09	0·10	0	Normal.
XXIV	35	F.	November, 1931.	January, 1932.	0·10	0·15	0·18	0·12	0·09	0	Normal.
XXVI	24	F.	October, 1931.	January 1932.	0·10	0·14	0·16	0·12	0·10	0	Normal.
XXVIII	38	M.	June, 1928.	January, 1930.	0·12	0·18	0·21	0·21	0·18	+	
				June, 1930.	0·24					+	On insulin, 15 units, twice a day.
				April, 1931.	0·32					+	On insulin, 30 units, twice a day.
XXIX	48	F.	January, 1932.	January, 1932.	0·10	0·15	0·19	0·21	0·23	+	1 week after drainage.

CASE XXVIII.—A male, aged thirty-eight years, had been under observation for two years before the development of pancreatitis because of chronic alcoholism. No evidence of *diabetes mellitus* was found during this period. In June, 1928, he developed acute pancreatitis and was operated upon. He remained well after the operation until January, 1930, when he commenced to lose weight and at this time he developed extreme thirst. Sugar was found in the urine and a tolerance test revealed a definite departure from the normal. Dietary treatment was commenced, but the subsequent course of the patient has been unsatisfactory because of his lack of cooperation in his treatment and because of frequent drinking bouts. Despite the use of insulin in gradually increasing amounts he has steadily gone down hill. His tolerance to carbohydrates is apparently decreasing, and the present insulin dosage is in the region of 70 units per day.

One other case is worthy of mention, although it does not illustrate a disturbance of tolerance in a recovered case.

CASE XXIX.—A female, aged forty-eight years, was admitted to hospital in January, 1932, with an acute pancreatitis. At operation it was found that the tail of the pancreas was affected. Although no sugar had been found in the urine on repeated examinations, a tolerance test revealed a definite impairment and glycosuria was present after the test. Her condition at operation was extremely grave and one week after the test was made she died.

It is problematical whether this patient would have developed *diabetes mellitus* had she recovered.

SUMMARY.

Eleven patients known to have suffered from acute pancreatitis have been investigated by means of the glucose tolerance test.

In eight cases the response to the test was normal.

In one case after a period of impaired tolerance, a normal response was obtained after a septic gall bladder was removed.

In one case after a period of impaired tolerance a normal type of response was obtained, but was associated with a condition of lowered renal threshold to sugar.

In one case a true *diabetes mellitus* apparently developed as a sequel to an acute pancreatitis eighteen months previously.

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ABNORMAL RETINAL CORRESPONDENCE: ITS IMPORTANCE IN THE TREATMENT AND PROGNOSIS OF CONCOMITANT SQUINT.

By T. A'B. TRAVERS.

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THE existence of anomalous forms of retinal correspondence has been a matter of common knowledge on the Continent for many years, but their great importance as regards the prognosis and treatment of squint does not appear to have been recognized by English and American authors. The German literature particularly contains numerous articles regarding the condition,[1][2][3][4] but we found no mention of it in the English or American works until Moncrieff,[5] in 1929, set out its salient features.

Before discussing abnormal or anomalous retinal correspondence, we shall refer briefly to normal correspondence.

It is well known that the eye localizes external objects by means of the impressions that it receives on the retina, as in Figure I. B, to the left of O, is interpreted as being along the line Bb, and O as being along the line OM.

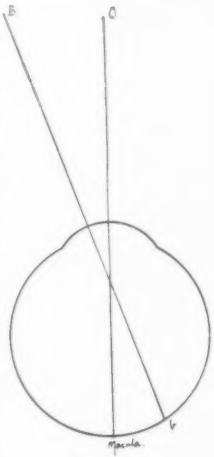

FIGURE I.

In Figure II we see the object O fixed by both eyes. Its absolute position is interpreted by the two eyes, as it is the only point that is common to the two lines OM and OM′. Similarly, B, to the right of O, is localized at B, the meeting point of Bb and Bb′.

This, briefly, is the usual description of the method by which the eyes localize the position of external objects. But the foregoing is only an incomplete description of the interpretation of direction and localization of points in space, for in the normal individual, associated with the impulses that come from the luminous points on the retina, there are proprioceptive impulses from the ocular muscles and from the head and neck, which modify the brain's interpretation of the simple visual impressions. As this means of localization of direction is not so self-evident as that previously mentioned, an illustration will be given.

If the eye fixes a light and then turns, say, from left to right, there will be a movement of luminous points on the retina from right to left. If the light is moved from left to right while the eyes remain still, an exactly similar movement of luminous points takes place, but in the first case the light is seen to stay still and in the second to move. Obviously the proprioceptive impulses have so modified the visual impulses that the correct interpretation is made by the brain.

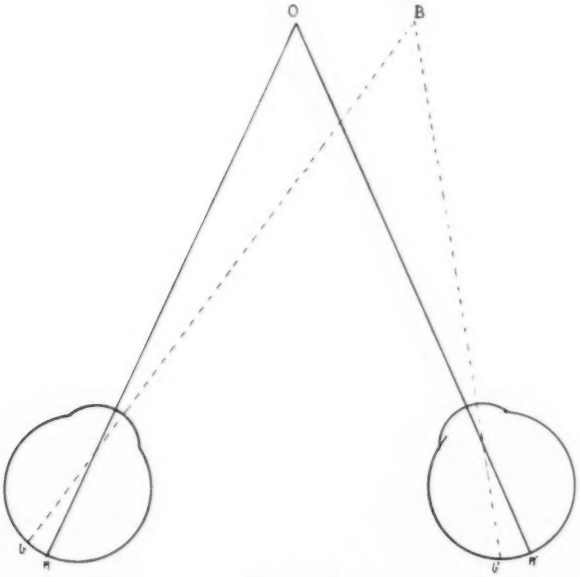

FIGURE II.

Again, if the eye be artificially moved, by the pressure of a finger, external objects are apparently seen to move. Here the lack of proprioceptive impulses to modify the visual impulses produces an illusion of movement.

There are numerous other examples of this modification of visual impulses, as, for example, the faulty localization of external objects by a person who has an ocular palsy, and the incorrect interpretation of the size of objects by persons whose eyes are under the influence of eserine or atropine.

Now the various factors which influence the interpretation of the simple visual impulses, be they proprioceptive impulses or an intangible function, such as memory or experience, have been classed together and called the "adjustment factor".[6]

So we may say that the interpretation by the brain of the direction in which an object lies is determined by: (i) The position of the luminous points on the retina, (ii) the "adjustment factor".

CORRESPONDING POINTS.

When the eyes fix an external object, both maculæ receive its image, and these two images are interpreted by the brain as single. The maculæ

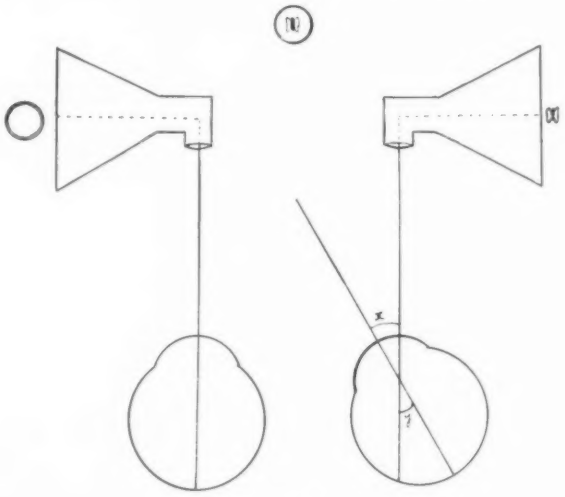

FIGURE III.

are said to be corresponding points and any other two points, whose simultaneous stimulation produces a unitary impression, are corresponding points.

The disposition of corresponding points around the two maculæ is usually considered to be a simple geometrical procedure. Thus a point two millimetres to the left of the right macula corresponds with one two millimetres to the left of the left macula, and so on.

That this is usually so is undoubted, but once again the precise relationship is not essential. A simple illustration of this fact is the fusion of stereoscopic pictures. Here the psychical blending of points not mathematically corresponding produces a unitary impression and is the basis of the perception of depth.

One may say that in normal correspondence the macula of the left eye corresponds with the macula of the right, and that the geometrical disposition of corresponding points around the two maculæ is approximately the same.

ABNORMAL CORRESPONDENCE.

In certain cases of squint the correspondence is so altered that the macula of one eye definitely corresponds with some point other than the macula of the other eye. When this has occurred, we say that an abnormal correspondence has developed.

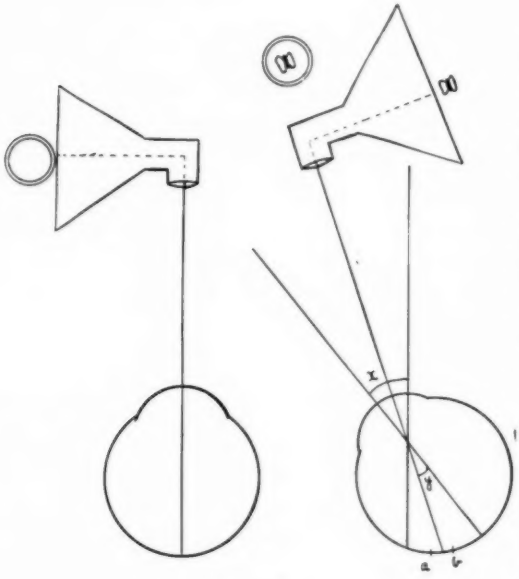

FIGURE IV.

There are two main types of abnormal correspondence which are represented in Figures III and IV. In Figure III the two eyes are seen looking into an amblyoscope which is representing two pictures, a butterfly and a net, for superposition. The patient in Figure III can "put the butterfly in the net" with the amblyoscope set as if the visual axes of the eyes were parallel, even though one eye is converged. This type of anomaly is called an harmonious abnormal correspondence,[7] as the angle of the anomaly y is equal to the angle of squint x. This variety of anomaly is very uncommon, and the usual form is the unharmonious abnormal correspondence,[7] represented in Figure IV.

In this type the butterfly will not usually go into the net as the suppression is too strong. It will appear to "jump" from side to side across the net, never actually appearing in it. At a, it would appear on the right of the net and on the left at b. This type is called unharmonious because the angle of the anomaly y is not the same as the angle of squint x. Actually y is always less than x.

Now, why does an abnormal correspondence develop? A seemingly rational explanation of the harmonious type is obvious. A child who had squinted all his life would avoid diplopia, and also the need for suppression, by learning to correlate the impulses from one macula and the other eccentric area. In this way there would be developed a crude form of binocular vision as, indeed, appears to occur in some cases.

But the unharmonious type, which is far the commoner of the two, is not so easy of explanation. A theory of its origin has been put

forward,[5] namely, that unharmonious types were at one time harmonious and have become unharmonious as the angle of the squint lessened. There is, however, very little to be said for this argument, as, if this were so, harmonious cases would not be the rarity that they are, and, in the majority of cases, there is seldom reliable evidence that the angle of squint has lessened. Again, if this theory were correct, the angle of the anomaly in the unharmonious types would be larger than the angle of squint, instead of the reverse, as is always the case.

We are suggesting that abnormal correspondence, whether harmonious or unharmonious, arises from the phenomenon known as "confusion",[8] the salient features of which will now be described.

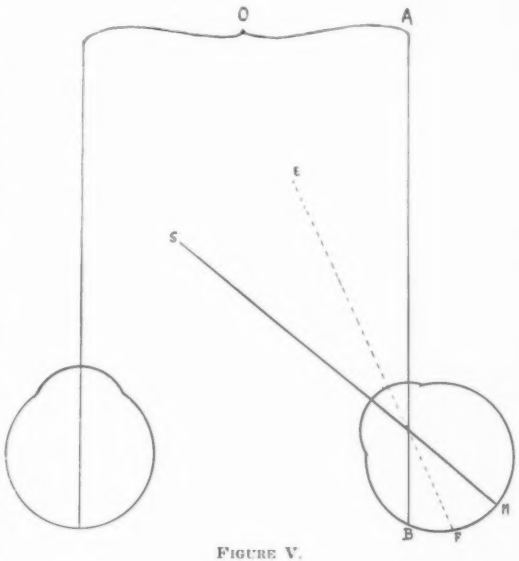

FIGURE V.

A man, whose eyes were normally straight with perfect binocular vision, developed, in certain circumstances, a divergence of one eye. When this occurred he usually had no diplopia, as the vision of the diverging eye was suppressed. But if the attention was directed to the objects seen by the diverging eye, the phenomenon of "confusion" occurred. If the attention was directed first to the object seen by one eye and then to the object seen by the other eye, each object was seen separately and in its correct direction objectively; but when seen simultaneously, there was an irresistible impression that the two objects were close together.

This illusion of approximation,[8] or "confusion", is presumably the result of the rivalry between the normal retinal correspondence on the one hand, which interprets the images on the two maculæ as being superimposed, and the knowledge, on the other hand, that the objects are far apart. While a "confusion" is in progress, there must be a dissociation of the adjustment factor between the two eyes.

It will be easily seen that a long-continued state of confusion will lead to a well-established abnormal correspondence.

There are two opposing factors—normal retinal correspondence and the inward certainty of knowledge and experience. This may be represented in Figure V.

Normal correspondence seeks to correlate the impulses from the fixing left macula with those arising from the right macula M. The patient, knowing the object O to be along AB, seeks to correlate the impulses from the left macula with those arising from B. A compromise is reached in which the correlation is between the left macula and F. Now, if these two conflicting forces are equal, F should be half-way between B and M. The stronger the normal correspondence, the nearer F is to M, and the weaker the normal correspondence, the more nearly does F approach B. Should F reach B the abnormal correspondence becomes harmonious. Thus the harmonious type suggests a very effectual suppression of old relationships and a very well established new mechanism.

A "confusion" is an alteration in the usual interpretation of direction by the brain when one eye squints. The interpretation of each eye separately is quite correct. It is a fault in the binocular interpretation of direction. It is, we suggest, essentially the same as an abnormal correspondence.

We have found that a very slight type of abnormal correspondence is demonstrable in many cases of squint, in that the angle of squint as measured by the Maddox rod is considerably less than that measured by the amblyoscope.

In this particular type, the abnormal correspondence vanishes as soon as the attention of both eyes is directed towards definite objects. In many of these cases there is no difficulty in the simultaneous perception and blending of binocular pictures, and depth perception may be demonstrable.

The above, we suggest, is the mechanism by which an abnormal retinal correspondence develops. Upon the varying strength of the two opposing factors—normal retinal correspondence and inward knowledge—will depend the squinter's binocular interpretation of direction.

At one end of the scale we have the very faint abnormality, only demonstrable by special methods, at the other the harmonious abnormal correspondence, indicating an almost complete suppression of normal relationships.

THE IMPORTANCE OF ABNORMAL CORRESPONDENCE IN TREATMENT AND PROGNOSIS.

One of the first steps to be taken in any case of squint should be to ascertain if the correspondence is normal, when two definite objects are presented simultaneously to the two maculæ. If, when this is done, the two test objects are seen far apart instead of superimposed, a well-established abnormal correspondence is proved, and this must be broken down if a cure is to be attained.

If it can be broken down by training, as it can in some cases, the outlook is much better, as then proper fusion may be established with its concomitant benefits.

Our practice at the Alfred Hospital is to train patients who have normal correspondence for a varying time, and then, if they do not appear likely to be cured with exercises alone, to operate.

We have found that those patients who have fusion before operation, do very well after operation; their eyes remain straight and even if the deviation be not entirely corrected at operation, the eyes commonly become straight in a few days or weeks afterwards. Stereoscopic vision is attained and a cure is complete.

The proper treatment of patients with an abnormal correspondence is still uncertain, but if the anomaly cannot quickly be broken down by training, we operate at once. Such patients often develop a convergence after operation, even if the eyes are put quite straight at operation. This is doubtless due to the eyes trying to attain their old and well-established method of working. Therefore, one should produce an over-correction in such cases at operation.

If the abnormality is not too deeply ingrained, the eyes will gradually develop normal correspondence in the course of time, but its attainment is often slow, and thus it seems best to operate early and so to prevent the further conditioning of an abnormal process.

From what has been written, it will be apparent that retinal correspondence is exceedingly important as regards prognosis.

Patients who have normal correspondence and fusion can be confidently expected to do very well. A cure with full stereoscopic vision can be reasonably anticipated.

A patient with abnormal correspondence has a much poorer prognosis. The more deeply ingrained the anomaly, the worse is the outlook for a cure and the greater the difficulties of operation.

SUMMARY.

1. A brief description of normal retinal correspondence and the salient features of abnormal retinal correspondence are given.

2. A theory of the origin of abnormal retinal correspondence is described.

3. The great importance of retinal correspondence in the prognosis and treatment of concomitant squint is emphasized.

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FRACTURES OF THE NECK OF THE FEMUR TREATED BY A FIBULAR BONE GRAFT.

By Basil Kilvington,
Honorary Surgeon, Melbourne Hospital.

Fractures of the femoral neck are probably the most troublesome of all fractures. The prognosis of those situated far out in the neck approaching the intertrochanteric type is more favourable than of those close to the head.

Sometimes we see impacted neck fractures, but those with loose ends, and definite displacement with shortening, and outward rotation, never give a satisfactory result with the ordinary treatment by splinting. The two causes of failure, I believe, are, first, that efficient reduction and immobilization are not obtained and, secondly, that weight bearing is permitted before bone union is firm.

Many of the patients with these fractures are too old and feeble to be treated properly, and the injury leads sooner or later to death as described in all the textbooks, but a surprising number (in fact, a majority of the patients) are able to stand the long period necessary for proper treatment.

The Whitman method of immobilization in plaster, with the shortening overcome and the outward rotation corrected, was a vast improvement on older methods, and the gone graft is really a variety of this method. When I was in Europe, Professor Nordenbos, of Amsterdam, was kind enough to demonstrate his graft method to me. The patient on whom he operated, was a man of sixty-six years, with syphilis of the spinal cord; and his fracture was subcapital. This was not an ideal patient. Professor Nordenbos has done 160 operations of this kind without a death and with very good results in all the cases I saw. Of course, he does not operate on very decrepid patients, and does not think operation necessary in the purely intertrochanteric type.

I recently showed a small series of patients with these fractures on whom I have operated as a demonstration before the members of the Royal Australasian College of Surgeons, and the following is the technique adopted.

The patient is anæsthetized with gas and oxygen, which give all the relaxation necessary, and is placed on an Ord's fracture apparatus. This is not essential, and Nordenbos uses a pelvis and shoulder elevator with a pelvis support strapped on to an ordinary table. Traction is made

by a powerful assistant by means of a clove hitch round the patient's ankle and continued to a belt round the assistant's waist. The pull is continued during the operation.

Three positions of the limb have to be studied.

1. Extension to get the limbs of equal length. Careful measurements should be made, as with the apparatus over-extension has been given with a disturbance of the measurements in inserting the graft. With over-extension the bone peg passes below the head, as is shown to have nearly taken place in one of the skiagrams in my own series.

2. Internal rotation of the whole limb to an angle of 12° to 15°.

3. Full abduction of the leg at the hip joint.

The fibular graft is now taken from the same side, about 12·5 centimetres (five inches) being used. This is freed from all muscular and fibrous attachments by scraping it clean with the scalpel. The incision to expose the femur is one 15·0 to 20·0 centimetres (six to eight inches) long lateral to the trochanter, going right to the bone. A one centimetre drill is now inserted for a distance of about 10 centimetres (four inches) (the exact length can be estimated by the X ray measurement, which is about one-fifth larger than that of the skeleton). It enters the outer surface of the shaft about one centimetre below the great trochanter, and passes horizontally and in a line directed to the opposite anterior superior iliac spine. When the region of the break is reached, a loss of resistance is easily appreciated and the drill is inserted only a short distance further. If it goes too far, it actually penetrates the articular cartilage of the femoral head and enters the hip joint. In my earlier cases I had an immediate X ray examination made, while the patient was under the anæsthetic, to make certain that the drill was in proper position, and on several occasions I found the track was not correct. With practice this is not necessary, and Nordenbos does not do it, and apparently all his grafts are in good position.

The two common errors are over-extension of the limb so that the fibular graft misses the femoral head or just impinges on the lower border of it, and driving the graft too far into the bone so that it approaches or actually enters the joint. The remedies for these are obvious. The fibula is hammered in the bored track, and I have used an ordinary thimble over the outer end to prevent splitting by the mallet. When the correct distance is obtained, the excess of fibular bone is clipped off. The wound is sewn up with catgut and the limb is at once put up in plaster. I have not included the sound leg in the plaster, but Nordenbos includes it as far as the knee. It is important to carry the plaster almost from the axilla to the toes on the injured side. On two occasions I merely extended the plaster to the ankle, but I found the whole leg rotated outwards slightly in the casing, and, as this is a bad fault, I should not do it in future.

The patient is kept in this plaster for three months, and it is found that he can be moved on to his sound side by supporting the injured

leg by a sling from the horizontal piece of the four-poster frame. This is important from the point of a hypostatic lung condition.

At the end of three months the plaster is removed; the patient bears no weight on the limb, but is given massage and is encouraged to perform all movements at the hip and knee joints. At the end of six months the leg should be quite capable of any movement. For the third period of three months I allow walking with the leg in a Thomas knee splint with heel callipers to take portion of the body weight from the hip, but Nordenbos allows walking at six months and does not use a Thomas splint.

In nine months the patient should have a strong leg with little or no shortening, and, above all, no outward rotation at the hip. The results have in my practice been superior to those obtained by any other treatment.

Most patients, except the very old and decrepid, will stand this operation, which is very little more trying than Whitman's plaster method of treatment.

Nordenbos has done 160 operations without, he tells me, a failure and with no mortality. I had one death in my own series from coronary occlusion four days after operation. The patient was a woman of fifty-seven years who had had many anginal attacks, and I operated on her with considerable misgiving, even though her physician assured me he thought the procedure a moderately safe one.

I believe that the sooner after the accident the operation is performed, the better (of course, allowing three or four days to elapse for recovery from shock). I do not feel so certain as to when to advise a pure plaster fixation and when to use a graft. In recent cases there is so little difference in the length of time taken that I prefer the graft. When three or four weeks have elapsed, the fractured ends have become more or less sclerosed and the neck partly absorbed, so that a graft is essential for any union at all.

When the fracture is of the intertrochanteric type there is a broad raw area for bone union, so that grafting is not essential, though it does no harm. My opinion is that the graft method is at the present time by far the best way of treating fractures high up in the neck of the femur.

ILLUSTRATIVE CASES.

The following is a *résumé* of the notes of some cases in which this method has been used.

CASE I.—N.T. was a female patient, aged sixty-five years. The fracture was of the pertrochanteric type with outward rotation and five centimetres (two inches) of shortening. Operation was performed on the third day after injury. The skiagrams (see Figures I and II) show the original fracture with the steel drill in position, and the graft with the leg in plaster.

CASE II.—M.R., a female patient of fifty-three years of age, was operated on eight weeks after injury. There were 3·75 centimetres (one and a half inches) of

FIGURE I.

FIGURE II.

shortening, with outward rotation and marked absorption of the neck. The skiagram shows the graft driven in with the thimble on the portion beyond the trochanter not yet cut off. The position is much improved, but the graft has been driven rather far in and is almost in the joint.

CASE III.—K.C., a female patient, aged sixty-one years, had a fracture high up in the neck with 1·8 centimetres (three-quarters of an inch) of shortening and marked eversion. Operation was performed six days after the accident. The skiagram shows the original injury, the drill is in position, but due to over-extension of the limb it passes just below the head of the femur. The drill was then inserted higher up and the graft was hammered in, but even now the fibula just lies in the lower part of the head and neck.

FIGURE III.

CASE IV.—M.F., aged fifty-seven years, has eversion and 1·8 centimetres (three-quarters of an inch) of shortening. She was operated on five days after injury, and the skiagram shows an excellent position with the drill in place. This patient is the one who died shortly after operation of coronary occlusion.

CASE V.—M.D., a female patient, aged fifty-three years, had 2·5 centimetres (one inch) of shortening, with eversion and marked absorption of the neck of the femur. X ray shows improved position. The graft has entered the lower part of the inner fragment.

Figure IV.

Figure V.

FIGURE VII.

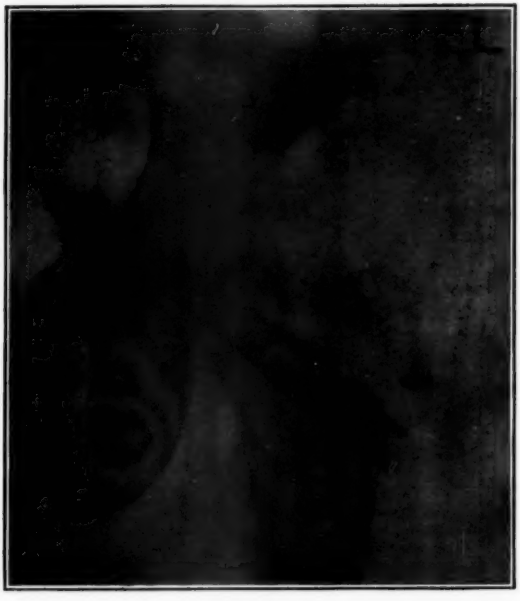

FIGURE VI.

CASE VI.—J.W., a male patient, aged sixty-eight years, sustained an injury four days previously, and the shortening was 1·25 centimetres (half an inch), with considerable eversion. The bone graft is shown with no shortening (see Figures III and IV). This fracture is so far out in the neck that it could have probably been treated by plaster alone.

CASE VII.—F.N., a female patient, aged fifty-nine years, was operated on nine days after the accident. The fracture was of the subcapital type, with 2·5 centimetres (one inch) of shortening and also external rotation (see Figure V). The whole bone is rarefied. The first drill barely hits the head of the femur, but the second is in good position (see Figure VI).

The skiagram of the fibular graft in position shows that the displacement has been well rectified (see Figure VII).

ACUTE OBSTRUCTION OF THE SMALL INTESTINE.

By B. T. Edye,
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In this short paper on acute obstruction of the small bowel I have confined my remarks, in the main, to mechanical obstructions occurring within the abdominal cavity. I have excluded intussusception, obstructions due to vascular lesions, all forms of strangulated hernia and paralytic ileus. In addition to drawing on my personal experience, I have reviewed the histories of the patients admitted to the Royal Prince Alfred Hospital over a period extending from 1922 to 1931. During this period there were 70 cases of the type mentioned, and in Table I they are classified according to the causes.

Table I.

Cause of Obstruction.	Males.	Females.	Total.
Adhesions and bands	20	36	56
Meckel's diverticulum	2	—	2
Other diverticula	2	—	2
Gall stone impaction	1	2	3
Food impaction	1	1	2
Carcinoma of the ileum	1	—	1
Constriction ring	—	1	1
Spasm of small bowel	1	1	2
Cause unknown	1	—	1
Total	29	41	70

It will be noticed that adhesions and bands are the commonest causes of obstruction, and that all other causes are relatively rare. Under the heading of "adhesions and bands" I have included all those obstructions associated with or following some inflammatory condition occurring within the abdomen, for example, appendicitis, salpingitis, diverticulitis *et cetera*, or adhesions following operations for such conditions. Obstructions from adhesions are more common in women than in men because of the inflammatory conditions to which the pelvic organs are prone. The appendix is a prolific source of trouble in this direction. In some instances the obstruction was due to adhesions which remained after an old attack of appendicitis. In others the obstruction occurred simultaneously with an attack, and in still others it occurred at various periods after operation for that disease.

There were two patients in whom a Meckel's diverticulum associated with adhesions or bands was the cause of obstruction. One patient was aged nine and the other twenty years, and both died. There were two other patients with diverticula of the small bowel; the diverticula were associated with adhesions, but did not correspond to Meckel's diverticulum. These recovered after operation. There were three examples of gall stone obstruction and all three patients died. I have operated once only for obstruction due to a gall stone. This was in a private patient and the result was satisfactory.

Carcinoma of the ileum accounted for obstruction and death in one patient. A constricting ring which followed the reduction of a strangulated hernia, was the cause in one instance, and the affected segment of bowel was resected with satisfactory result.

The symptoms of acute intestinal obstruction are often ascribed by the patient to the ingestion of some unusual food or drink, and the medical attendant, as well as the patient, is often not unwilling to accept the explanation. However, there are instances in which the ingested material is the direct cause of the obstruction.

One patient in this series had eaten a dozen or more oranges, the undigested pulp of which had become firmly impacted in the terminal foot of the ileum. The pulp was recovered at operation, but the patient died.

In another instance, manderin segments had been swallowed whole and the ileum refused to pass them. This patient recovered after operation.

In a third example, not belonging to the above series, quarters of an unripe pear which were bitten in two and swallowed, caused death by impaction in the terminal part of the ileum.

Amongst the histories was one of a young man who was feeling very hot and who endeavoured to cool himself by taking seven ice-creams and iced drinks in succession. He was still uncomfortably hot and about to have another iced drink when he collapsed. He was taken to hospital, where he was submitted to gastric lavage by the casualty surgeon who recovered most of the drinks. However, symptoms suggestive of intestinal obstruction supervened, and he was operated upon and found to have dilatation and injection of the whole of the small bowel, with the exception of the terminal portion of the ileum, which was in a state of spasmodic contraction. Jejunostomy was performed and he recovered.

There was one other example of obstruction in which spasm of the ileum appeared to be the cause.

The patient in this instance was a woman, aged seventy years, who had been suffering for four days with abdominal pain and vomiting. Jejunostomy was performed, but she died next day.

In the above series there was one very unusual case of obstruction.

The patient, a woman, aged thirty-five years, was known to have very large polycystic kidneys. Some of the cysts of the lower pole of the left kidney, where it projected into the abdomen, had become acutely inflamed. The inflammation involved the peritoneum and some adjacent coils of small intestine, leading to obstruction. The coils of bowel were freed and the inflamed cysts drained by the lumbar route with good effect.

It is interesting to note that the colon is seldom acutely obstructed by adhesions. Because of this I consider it an advantage, after any operation, to adjust the colon and caecum in addition to omentum,

whenever possible, over denuded or oversewn areas in such a manner as to exclude the small bowel.

It is well to remember that when adhesions or bands cause obstruction, the bowel may be affected in more than one place.

In one instance I was asked to see a child who had been operated upon for obstruction which occurred during convalescence after removal of a gangrenous appendix. The symptoms of obstruction had persisted, and at a further operation other obstructed coils of small bowel were relieved and the child recovered.

There are occasions when the suspected cause is not the one found.

I recently operated on a woman, and repaired a large incisional sub-umbilical hernia in the sac of which small bowel was firmly adherent. Three days later symptoms of acute obstruction appeared. It was reasoned that coils of small bowel freed at operation had become obstructed. No time was lost, as exploration in the presence of distension would have been exceedingly difficult. The abdomen was opened in such a manner as not to interfere with the repaired hernia and the obstruction was found to be due to a short fibrous band in the floor of the pelvis.

In another instance obstruction occurred some ten days after operation for right inguinal hernia. The cause was the incarceration of small bowel in a large paraduodenal pouch, together with the strangling effect of a tight fibrous band.

In yet another instance obstruction of the small bowel occurred nine days after cholecystectomy and exploration of the common bile duct. The patient was a man and the onset of his illness was marked by violent abdominal pain, more severe than any pain he had previously suffered. He was operated upon within a few hours and the obstruction was found to be due to the kinking of the ileum thirty centimetres (one foot) above the caecum by an adhesion due to the organization of a fragment of blood clot which must have escaped during the previous operation.

And here I might stress the importance of controlling even the slightest hæmorrhage before closing the abdomen after any operation. Blood clot is partially absorbed and partially organized to form fibroblastic tissue. This young fibrous tissue contracts and leads to the formation of adhesions and bands, always a potential danger.

The recognition of acute intestinal obstruction is often delayed because of the occurrence of some unusual factor. The following is a brief account of such confusion.

A child suffered from recurring attacks of acidæmia associated with abdominal pain. Finally, an attack occurred in which the pain did not subside, but culminated in operation for perforative appendicitis. Some days later, pain and vomiting recurred together with the appearance of acetone in the urine. Again acidæmia was considered as an explanation, but as the symptoms persisted in spite of appropriate treatment, obstruction from inflammation and adhesions was diagnosed and ileostomy performed. Relief was immediate and recovery complete without further interference.

With regard to the division of bands, I once had the unpleasant experience, after the division of a short tight band which was attached at one extremity to the antimesenteric border of the ileum, of being confronted by the sudden and uncontrollable escape of the contents of the bowel into the abdomen. The explanation was the division of a narrow diverticulum of the ileum due to the traction of the band. The patient died mainly as the result of the shock from the sudden flooding of the peritoneal cavity by the irritating small bowel contents.

I remember another instance when the patient died almost at once; the abdomen had been opened and the site of obstruction searched for rather too hastily. There was a sudden flooding with fæculent fluid, due to the tearing of the ileum where it was constricted in an unrecognized femoral hernia.

The diagnosis of acute intestinal obstruction depends mainly on a careful history of the onset. In all the histories that I have examined, severe abdominal pain was almost invariably the first symptom. The pain was usually recurring and colicky. There was vomiting, and there was also some difficulty with the bowels. I regard the pain as the most important single fact, and if I can satisfy myself that the pain has been severe, I consider the patient is suffering from a surgical condition: and if, in addition, I can exclude biliary and renal colic, I seldom hesitate to recommend operation. If the patient is seen soon after the onset, but between the attacks of pain, the clinical findings may be normal, that is, the patient may be lying comfortably in bed without distension or tenderness or disturbance of pulse, respiration or temperature.

One afternoon I examined a young man in such a quiescent period, when suddenly he was seized with pain. Unfortunately, I was not convinced that it was genuine, and early next morning when I saw him he was moribund. At autopsy a strangled loop of small bowel was discovered.

A similar chain of events may occur during convalescence from such an operation as appendicectomy. The patient, who perhaps otherwise is progressing well, has an attack of abdominal pain and vomits. When the patient is examined there may be nothing unusual to note. Soon afterwards the pain and vomiting recur. In such circumstances it is generally wise not to wait any longer, but to prepare for appropriate operative treatment.

The nature of the vomit in small bowel obstruction is instructive. Bowlfuls are brought up, and the vomit, after the stomach is emptied, consists of bile-stained fluid. Later it becomes yellowish in colour and has a fæculent odour, the characters being exactly those of the contents of the distended small bowel, which may later be evacuated by enterostomy.

The disturbance of the function of the small bowel in respect to defæcation and the efforts to secure a satisfactory result are apt to be misleading. The colon may be loaded at the moment the small bowel becomes obstructed; there may be a normal evacuation or an enema may give more or less satisfaction. It is only when the patient is under trained supervision that the report on the results of the enemata are reliable. Usually untrained people take no note of the passage of flatus and yet the passage of flatus is of greater importance than the passage of fæces, or, in other words, if flatus is passed, with or without the assistance of enemata, we can be certain that the patient is not suffering from acute intestinal obstruction. Too often too much time

is wasted awaiting the result of repeated enemata, while other symptoms, particularly pain, are broadcasting the information we seek.

Distension is generally regarded as a late symptom, but there is some degree of it from the very beginning. However, because we have no knowledge of the normal contour of the patient's abdomen, we are unable to be sure of its presence. There is a sign which often gives valuable and conclusive information before distension is otherwise recognizable; I mean splashing or succussion. This sign should always be sought for over the small bowel area in suspected obstruction. If I discover it in an otherwise difficult case, I have no hesitation in completing the diagnosis and recommending operation.

Abdominal tenderness of varying degrees, and even rigidity, were frequently mentioned in the Royal Prince Alfred Hospital histories, and it must be admitted that their presence adds to the difficulty of differential diagnosis. Occasionally a palpable abdominal mass was described and such a mass usually consists of strangled coils of bowel distended with fluid. I can call to mind two instances in which the swelling simulated the position and outline of a distended bladder. In each the patient appeared unable to micturate and, as a catheter also failed to withdraw urine, suprapubic puncture was performed with disconcerting result. The confusion arose because of the suppression of renal secretion due to the dehydration of advanced intestinal obstruction.

Before leaving the subject of diagnosis, I should like to draw attention to the difficulty which is sometimes caused by obstructive lesions of the renal pelvis or ureter. A person suffering from such a condition may be seized with severe abdominal pain, not necessarily typical of renal colic. He vomits persistently, there is difficulty in obtaining a satisfactory action of the bowels and slight distension is detected. The whole of one's attention is apt to be focused on the gastro-intestinal symptoms and consequently the unfortunate patient is plied with enemata and even with aperients, which he usually vomits.

I recently saw a young man whose attack began after a violent bout of sea-sickness. He suffered for a week and finally passed a small renal calculus. An X ray examination showing the abdomen filled with coils of distended bowel, strongly favoured the diagnosis of intestinal obstruction. However, the patient throughout the course of his attack passed flatus, and this fact alone excluded a diagnosis of intestinal obstruction.

I suppose everyone present has met with young patients who have suffered from attacks suggestive of some gastro-intestinal disturbance in which the ultimate cause has been shown to be repeated attacks of acute hydronephrosis due to kinking of the ureter by an abnormal renal artery or some similar defect.

The reflex ileus associated with acute cholecystitis and acute pancreatitis is most confusing and frequently leads to a diagnosis of mechanical obstruction. A reliable nurse can give valuable assistance in solving the problem.

Among the seventy patients in the Royal Prince Alfred Hospital series there were twenty-three deaths, giving a percentage of 32·8 (see Table II). Four of the patients died without operation, three being *in extremis* when admitted, whilst the fourth was left too long before the diagnosis was made. Of course, several of the patients who died were in a serious condition when submitted to operation. The length of time that elapsed from the onset of symptoms to the operation in these patients varied from eighteen hours to nine days. What a difference there would be in mortality figures if the diagnosis could be settled within twelve hours! This should be possible, seeing that urgent pain calling for active treatment is usually the first and main symptom. The actual cause or causes of death in intestinal obstruction has been the subject of considerable investigation, but it is a problem the solution of which the surgeon must of necessity leave to the bacteriologist and biochemist. These researches have evolved new methods of treatment for the toxæmias of intestinal obstruction, for example, the use of hypertonic saline solutions to restore the chlorides and fluids, gas-gangrene antiserum to combat the toxins of the *Bacillus welchii*. None of the

TABLE II.

Cause of Obstruction.	Males.	Females.	Total.
Adhesions and bands	7	7	14
Meckel's diverticulum	2	—	2
Other diverticula	—	—	—
Gall stone impaction	1	2	3
Food impaction	1	—	1
Carcinoma of the ileum	1	—	1
Constriction ring	—	—	—
Spasm of small bowel	—	1	1
Cause unknown	1	—	1
Total	13	10	23

methods, however, can repair the damage done by failure to operate early. The suffering and anxiety associated with delayed treatment are in marked contrast to the smooth convalescence which follows early and satisfactory operation. This aspect should weigh with the medical attendant in coming to a decision and even tempt him to risk an unnecessary operation.

A general survey of the treatment adopted at the Royal Prince Alfred Hospital showed that elimination of the obstructing agent was all that was done at operation in the great majority of cases. Evacuation of the distended bowel, followed by closure, was mentioned in seven cases, ileostomy in three cases and jejunostomy in two cases. Ileo-ileostomy was performed once and anastomosis between the ileum and the transverse colon five times. Resection was mentioned three times, twice for gangrene and once for removal of a constricting ring. In

the after-treatment, normal saline solution, with or without glucose, was commonly administered by various routes. Hypertonic saline solutions had not often been used and gas-gangrene antiserum had been given only to occasional patients according to the enthusiasm of the surgeon in charge.

My own practice in early cases is simply to remove the cause of the obstruction. In all other cases in which operation can be attempted, I relieve the obstruction at once if the site is easily located, and then perform enterostomy by fixing a long piece of narrow tubing in the distended ileum with two purse-string sutures. After emptying the bowel as far as possible, using a "sucker" if available, I bring the enterostomy tube through a stab wound at a convenient distance from the main incision, the tube is drawn through until the bowel is in contact with the parietal peritoneum, and is secured to the skin with a

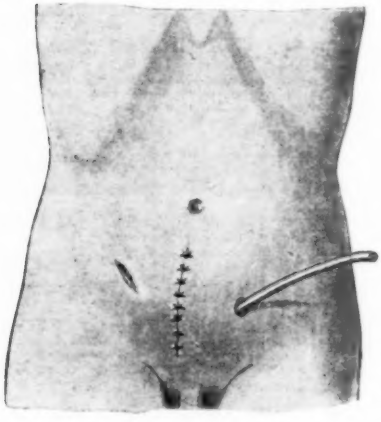

FIGURE I. The original aseptic wound is shown on the right. The exploratory incision is in the centre and the enterostomy tube drawn through a stab wound is on the left.

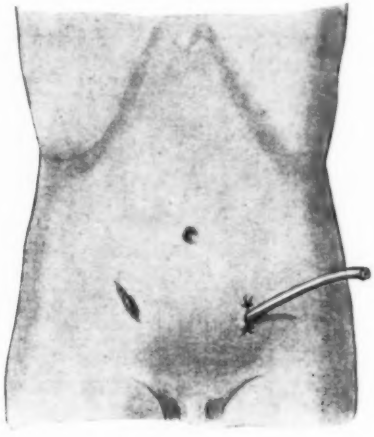

FIGURE II.
FIGURE II. The original septic wound is shown on the right. The incision for enterostomy with the tube in position is on the left.

silkworm gut stitch. If the enterostomy tube is brought through the main incision, delayed healing from infection is very apt to take place. The enterostomy tube is kept in position until the bowels act normally. After its removal the opening closes quickly. If the search for the site of obstruction is hindered by dilated coils, I immediately perform enterostomy and evacuate a quantity of the bowel contents before proceeding.

Preliminary gastric lavage is recommended and attempted in all cases, especially if a general anæsthetic is contemplated. The adminis-

tration of ether, even to patients who have not vomited to any great extent, may precipitate such an attack as to cause death from asphyxia.

In post-operative obstruction, where there is no sepsis, the procedures above described are followed (see Figure I), but if there is a septic wound, such as may follow operations for suppurative or gangrenous appendicitis, the infected area is left entirely undisturbed and simple enterostomy is performed at a site well away from it, that is, usually through the lowe rportion of the left rectus (see Figure II). In the latter type, as the inflammatory process subsides the bowel as a rule recovers its function. The enterostomy is then dispensed with.

Everyone is sadly familiar with the progressive peritonitis which sometimes follows appendicitis and with the difficulty of deciding whether further operative treatment should be advised or not. We know that the peritonitis is gradually causing paralysis of the bowel; we also feel that there is some degree of mechanical obstruction. In such cases simple enterostomy under local anæsthesia through the left rectus may give relief, while it will at least do no harm.

With regard to after-treatment, when the obstruction is relieved in the early stages, no special methods are instituted. In late cases, saline solution, preferably hypertonic, with glucose is given freely, intravenously or *per rectum*. I also administer gas-gangrene antiserum, but have not used it sufficiently to form an independent opinion of its value.

THE TREATMENT OF PERFORATED PEPTIC ULCERS.

By HENRY SEARBY,
Melbourne.

THE purpose of this paper is to present the results in a series of fifty-three consecutive unselected cases of perforated ulcer in patients on whom I have operated during the past five years. The age of the youngest patient in the series was nineteen and that of the oldest sixty-three. Three patients died, a mortality rate of 5·7%.

In Table I is given a list of mortality rates selected from the literature of the past five years. In most of the series different surgeons

TABLE I.

Author.	Number of Cases.	Mortality.
Bager (Sweden)	1,767	32·8%
Williams and Walsh (Liverpool)	158	30·0%
Fleming (review of literature)	1,000	25·0%
Judine (France)	195	24·0%
Urrutia (America)	34*	17·6%
Grey Turner (Newcastle) ..	157*	14·64%
Bryce (Manchester)	125*	13·0%
Present series	53*	5·7%

* "Personal".

have operated, but the figures of Urrutia, Bryce and Grey Turner are those of "personal" cases. All Bryce's patients were treated by infolding the ulcer, with or without drainage of the pelvis.

The mortality rate, as is well known, increases rapidly with delay in operating. It also rises rapidly after the age of fifty or thereabouts. In Tables II and III are shown the mortality rates in the present series

TABLE II.

Showing Results in Fifty-three Cases According to Time of Operation after Perforation.

Time of Operation After Perforation.	Number of Patients.	Number of Deaths.	Mortality.
Within 6 hours	19	0	0%
6 to 12 hours	24	1	4%
Later than 12 hours ..	10	2	20%

of fifty-three cases, both as regards time after perforation and as regards age.

Two of the three deaths in this series occurred in cases of thirty and thirty-six hours' duration respectively, from general peritonitis which was already well established at the time of operation. The other death was that of a man of fifty-two, who was operated on nine hours after perforation and who later developed pelvic and subphrenic abscesses. These were drained, but death followed about ten days later and *post mortem* examination revealed collections of pus between the coils of small bowel.

At operation two types of perforated ulcer can be recognized: (i) A type in which there are a marked inflammatory condition, a large area of induration, deposits of lymph in the vicinity and, usually, definite general gastritis. (ii) A type in which there is a small callous ulcer with little or no recent inflammatory change round it. In no case was perforation due to an "acute" ulcer.

TABLE III.

Showing Results in Fifty-three Cases According to Age.

Age.	Number of Patients.	Number of Deaths.	Mortality.
Under 50 years	36	1	3%
Over 50 years	17	2	12%

Bearing on the exact operative treatment I wish to emphasize five facts:

1. Closure depends for a short time only on the integrity of the sutures, irrespective of how they are inserted. Thereafter it is dependent upon the peritonealization of the infolded area. If the edges are sewn firmly together with through and through sutures, for example, union does not occur in this indurated infected tissue as it does in, say, a gastro-jejunostomy. The subsequent changes in the muscular and mucous coats depend on factors other than the method of oversewing. The authority for these statements has been obtained from the *post mortem* room and from the appearances seen at subsequent operations.

2. Leakage from an oversewn ulcer practically never occurs. (No such case has been seen at the Melbourne Hospital for the past twelve years.) This is because peritonealization occurs so rapidly. Hence a simple method of closure is just as good as an elaborate one.

3. Constriction of the lumen of the stomach or duodenum cannot occur in sufficient degree to matter even when large ulcers are infolded. Newton and Johnston[1] showed seventeen years ago that it was possible in dogs to suture the upper and lower borders of the duodenum together without causing symptoms. In perforated ulcers the sutures rapidly cut through and the lumen is reestablished.

4. Stomach and duodenal contents, in most cases, are not sterile. Of the fatal cases of perforation, over 50% are due to general peritonitis.

5. A tube placed to the bottom of the pelvis and brought out suprapubically to allow the escape of bacteria-laden fluid must not be considered as drainage of an infected peritoneal cavity, but merely as a good method of removing fluid which usually contains pathogenic organisms.

Although from first principles there is to my mind no question of what constitutes the correct operation, difference of opinion still exists. The ideal method of treating chronic peptic ulcer has not yet been achieved, and in the controversy which continues over the treatment of perforated ulcers, the treatment of the perforation is in many cases confused with the treatment of the ulcer. Treatment of the ulcer by surgical means is not an urgent matter; treatment of the perforation is. The least operative procedure which will convert the perforated ulcer into a non-perforated ulcer, which in its turn can be dealt with, is correct. This procedure consists of oversewing the ulcer.

Gastro-jejunostomy is not necessary because of the danger of narrowing the lumen nor because of the danger of leakage from the oversewn ulcer. The operation of gastro-jejunostomy carries a definite mortality even in non-perforated ulcers, and is not always the correct operation for non-perforated ulcers in the same position. The exact position of a perforated ulcer is frequently very difficult to determine, and I do not think it justifiable to disturb any protective lymph or adhesions to find out.

After the diagnosis is made, operation should be performed as soon as the patient can be prepared. Although delay up to about six hours does not appear to matter greatly, each hour saved means less leakage and less chance of peritonitis. Preparation consists of the administration of 0·015 gramme (one-quarter grain) of morphine, shaving, cleaning and painting the skin. The mortality increases so much with delay that operation should be performed as soon as possible, even under disadvantageous conditions, and a knowledge of the correct technical procedure should be in possession of all who are practising in isolated districts.

The incision should be made 2·5 centimetres (one inch) to the right of the mid-line, reaching to within 2·5 centimetres of the costal margin and should be long enough to allow the hand to be inserted in case a suprapubic stab wound has to be made. The rectus muscle should be dissected from its sheath and retracted laterally; and at this stage the skin edges should be protected well with towels, because the fluid which escapes when the peritoneal cavity is opened, may cause digestion of the wound and delay healing, and may perhaps also lead to a hernia. The body of the stomach is withdrawn as soon as the peritoneal cavity is opened, and held firmly between packs by the assistant. No other retraction is necessary. Two "Z" sutures of number "0" chromicized catgut are used to close the perforation. The first of these is placed beyond the indurated area (otherwise infolding is impossible in many

cases) and may, if necessary, grip the muscular as well as the serous coat. The second grips the serous coat only. During these steps the suction pump is used to clear the field and to remove as much fluid as possible. If drainage is decided upon, the left hand is put down to the suprapubic area, an opening is made and a tube with a window 1·25 centimetres (half an inch) from its end is placed so as to reach the bottom of the pelvis. The wound is then closed in layers and a collapsible tubular rubber drain is inserted down to the rectus sheath in case some of the escaped fluid has reached the fat.

Ethylene and oxygen is the most suitable anæsthetic, but if ether given by the open method is used, it is my custom to stop the administration as soon as the posterior rectus sheath is closed. In this way the rest of the suturing helps to wake the patient, who can then be placed in a sitting position in bed sooner than otherwise. The operation should not take longer than thirty minutes, and with easily accessible ulcers it can be completed in less than half this time.

When the patient is put back to bed, he receives one pint of saline solution by the rectum; this is repeated in two hours' time, and every four hours during the first twenty-four hours. Sips of water are allowed as soon as the patient is conscious, equal parts of milk and water after twelve hours, Benger's food in addition after thirty-six hours, and then the diet is gradually increased by the addition of custards, jellies, minced boiled fish and meat and bread and butter. I have found that during the first ten days alkalis are not only unnecessary (almost certainly there is not any hyperacidity on account of the accompanying gastritis), but very often distasteful to the patient.

The treatment in all cases has consisted of closure of the ulcer, and in all but seven drainage of the pelvis. The drain tube is removed after twenty-four to thirty-six hours. In one case the track formed by the tube resulted in obstruction of the ileum, necessitating a second operation ten days after the perforation had occurred, but in no other cases have any abdominal complications followed.

REFERENCE.

[1] Alan Newton and W. W. S. Johnston: "A Preliminary Note on the Treatment of Perforated Duodenal Ulcer", *The Medical Journal of Australia*, November 27, 1915, page 507.

Surgical Technique.

FRACTURE EXTENSION SYSTEMS: COMMON ERRORS IN THEIR ERECTION.

By Charles Gale,
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Very little of the literature concerning fractures gives much information as to the technique of the actual application of the more commonly used methods of extension and counter-extension. Bitter experience as a junior house surgeon, observation as senior house surgeon of similar experiences on the part of contemporary junior house surgeons, and discussion with various senior surgeons, have taught the writer many of the common mistakes liable to be made in the actual putting up of fractures.

The object of this paper is to describe these mistakes (which recur with monotonous regularity with each succeeding group of house surgeons), and to enumerate various suggestions concerning their prevention and cure. Unless otherwise stated, the description refers to the lower limb as the example.

The Extension of Fractures.

An extending force can be applied to a limb either by (i) attaching it to the skin, or (ii) attaching it to a bone. Let us confine our attention to the first of these.

Three materials for transmitting prolonged extension are in common use at the present time, namely, adhesive strapping; glue, usually in the form of Sinclair's glue; plaster casing.

Adhesive Strapping.

Adhesive strapping has several disadvantages. First of all it tends to slip over the skin surface. No matter how efficiently it is applied, an initial slip of one to three centimetres usually occurs during the first few days. Thereafter it may or may not continue to slip steadily until it may eventually become completely detached from the limb. The degree and rapidity of slip depend upon the carefulness of application, the quality of the strapping, the weather, the area of skin surface utilized, and the amount of the extending force. Even under the most favourable circumstances, however, there is usually some slipping, and this brings us to the second disadvantage, namely, cutting into the tissues.

The degree of cutting in depends upon the degree of the slipping and the tightness of application of the circular or oblique strips of strapping that are generally used. Many times the writer has applied strapping extension to a leg while erecting a Hamilton Russell extension, and, immediately after completing the procedure, has been very pleased with its appearance and satisfied that the bony prominences have been sufficiently protected with padding. Then a few days to a week later it is found that the oblique or horizontal bands have slipped distally and are cutting in at various points, particularly at the upper border of the patella, the upper portion of the malleoli, the instep, and the heel just

proximal to the attachment of the *tendo Achillis*. If the strapping *et cetera* is enclosed in a casing of cotton wool and bandages, and if the patient is uncomplaining, or if he regards the pain of cutting in as a necessary concomitant of the treatment, then a pressure sore may be discovered only when it is well established. This is unfortunately a common experience. Because of these disadvantages the writer has discarded strapping in favour of glue, but it is suggested that the strapping enthusiast should observe the following points.

1. To minimize slipping, first use the best strapping obtainable and, secondly, plan carefully the distribution of the circular and oblique bands that are to be used for maintaining the main longitudinal extension bands in apposition with the limb. A useful manœuvre is exemplified by a description of the method of attaching the lateral main longitudinal strapping bands to the outer side of the right lower extremity in erecting a Hamilton Russell extension. The operator stands facing the outer aspect of the right lower limb (that is, the patient's feet on his right). The strapping is first attached to the skin lateral to the knee joint, then, with the left hand, the proximal end of the strapping is drawn taut towards the patient's hip, while the right thumb and forefinger straddle the as yet unstuck proximal end of the strapping. The right thumb and forefinger then drag the loose skin and subcutaneous tissue distally as far as possible, after which the taut strapping is pressed on to the wrinkled skin. The attachment of the remaining strapping is carried out with the use of the same manœuvre. In a few days it is found that the slight initial slip does occur, but that the strapping remains taut and evenly attached to the skin.

Thirdly, do not, prior to attaching the strapping, clean the skin surface with any solution containing soap.

Fourthly, the adhesive property of the strapping is increased by swabbing the adhesive surface lightly with chloroform immediately prior to its application. Ether does not have this effect, but is excellent for removing strapping.

2. To prevent cutting in, first distribute all circular and oblique bands in such a way as to allow for the inevitable initial slip. For example, if it is decided to apply a circular band above the ankle or above the knee, make certain that its lower edge is at least five centimetres (two inches) proximal to the upper portion of the malleoli and patella respectively. Secondly, pad all prominences (bony or otherwise) where there is a possibility of cutting in taking place. Thirdly, examine all portions of the limb at frequent regular intervals quite irrespective of whether the patient is complaining or not.

Glue.

Glue is most efficient, providing it is of a suitable formula and is applied with meticulous attention to certain details. Failure of glued extensions is usually due to failure of attention to these details.

The arguments advanced against glue are: (i) It blisters the skin. (ii) It flakes off the skin.

Blistering is due to either: (*a*) the glue, (*b*) the nature of the skin and underlying tissues, (*c*) the amount of pull exerted per square inch of skin surface.

The Glue.—It is often said that blistering is due to the glue being too hot. Provided the skin and underlying tissues are healthy, and that the pull per square inch is not excessive, it is doubtful if hot glue *per se* does cause blistering. At one time, the writer was employed for eight hours a day over a period of months glueing cardboard stiffeners into leather kitbags. Many times the forearms, and occasionally the face, were splashed with glue much hotter than that usually used in applying extensions. At no time did anything more than an erythema result. The chemical constitution of the glue is occasionally blamed; against this the same example is quoted.

The Skin and Underlying Tissues.—If the tissues are distended with either extravasated blood or with œdema resulting from interference with the vascular mechanism due to marked trauma at a more proximal level, then blistering is more apt to occur and may be expected.

The Pull.—The smaller the skin surface utilized and the greater the pull used, the greater the tendency to blistering. Corlette[1] suggests that the total pull should not exceed twenty pounds, while Sinclair[2] advocates a maximum of twelve to fifteen. So, to prevent blistering, test the temperature of the glue on the back of the hand or forearm, use glue of one of the accepted formulæ, for example, Sinclair's, and distribute the pull over as large an area as possible.

If the tissues of the limb present unfavourable features, then glue may still be used on the offchance that a favourable result may occur, or else another method of attaching the pull may be utilized. Even if blistering does occur, it often does not impair the efficiency of the apparatus, the remaining unblistered skin being sufficient to take up the strain. It is uncommon for the blisters to show evidence of infection, but if this should occur it is rapidly controlled by a few applications of methylated spirits.

"Flaking-off" of the glued strips from the underlying skin is most frequently due to bad technique in the application of the glue. The errors in technique resulting in a tendency to flake off are: (*a*) applying the glue too thickly, (*b*) leaving the edges of the glued strips exposed to accidental knocks, these tending to peel them off the underlying skin.

To apply glue efficiently the following technique is suggested. The apparatus consists of glue of a suitable formula, a glue pot, a suitable brush and a piece of rod iron.

At the Alfred Hospital, Melbourne, glue is made according to the following formula:

Commercial glue "pearls": 50 parts.
Water: 75 parts.
Glycerine: 4 parts.
Menthol: 1 part.

The glue is allowed to soak in the water for one hour, and is then heated gently until the glue is melted. The menthol is dissolved in the glycerine and the solution is mixed with the melted glue. Pour into a flat dish and cut into cakes when set. Store in airtight tins.

Other formulæ are suggested by Sinclair,[3] Page and Bristow,[4] and others.

An ordinary commercial one-half to three-quarter pint glue pot is obtainable for a few shillings at any fairly large ironmongery. The glue pot consists of a large iron pot (with a handle of the water-bucket type), which is a third part filled with water. Fitting into this is a smaller similar pot into which the glue cakes are placed. There is no necessity, as is sometimes supposed, to protect the glue from steam. The pot is then heated until the water comes to the boil, after which it is allowed to simmer until the glue is completely melted, at which stage it is ready for use as follows:

A suitable brush, such as a painter's brush with the hair portion one and a half inches wide by one-quarter to three-eights of an inch thick by one and a half inches long, is dipped in the hot water in the larger pot so as to warm the hairs. It is then dipped into the glue and used as a stirrer, being wiped from time to time upon a piece of iron rod, approximately one-quarter of an inch in diameter by six to eight inches long, so as to fill the hairs with warm liquid glue. The glue is then diluted to a suitable thin consistency by taking a series of dips with the brush into the hot water and then mixing it with the glue. There is a fallacy that water should not be mixed with the glue. This is entirely wrong. Glue cakes when melted are often too thick for satisfactory working, and must then be diluted. Excessive use of the brush as a stirrer is to be avoided, as it is apt to make the glue frothy and unsuitable.

The unshaved skin is cleaned first with ether and then with soap and hot water containing four drachms of sodium bicarbonate to the pint. The dry skin is then warmed. A brushful of glue, with the excess rubbed off on the stirrer, is then (after making certain that it is not too hot) rubbed sparingly into a fairly small area of skin, finishing with a sweep of the brush in a direction opposite to that in which the extension will pull. The material (flannel or gauze bandage) is then carefully laid upon the glued skin and pressed into position. If the narrow

flannel strips as used with Sinclair's footpiece are being applied, it is recommended to press them into position with strokes of the pulp of the forefingers in opposite directions, from the centre outwards in a direction transverse to the long axis of the strip. Stroking parallel to the long axis is apt to lift the strip. If the glue is of the correct consistency, it should just squeeze through some of the interstices of the flannel and appear on its superficial surface.

If gauze bandage is being used (for example, gauze or butter muslin folded to six thicknesses so as to form a bandage two and a half to three inches wide), it is very carefully placed in position and glued into place by the stroking motion described above. It is then usually found that only two or three of the thicknesses are stuck down, in which case the superficial half is carefully folded back as in opening a book, and a fresh layer of glue carefully applied to the superficial surface of the layers already stuck in position. Particular care is taken that the brush contaminated with cooled tacky thick glue is first washed thoroughly in the glue pot water, and then well stirred with the glue, so that it is now charged with warm glue of the correct consistency. The commonest mistake in using glue is to dip the brush charged with cooled thick glue into the glue pot and then to rub into the part the mixture of warm thin glue *plus* the underlying cooled thick mess.

Inevitably the result will be bad. The superficial layers of gauze are now folded back into position and stroked into contact. A bandage is then evenly and moderately firmly bandaged over all. This serves not so much to keep the strips in apposition with the skin as to prevent accidental lifting of the edge of the glued material by contact with bedclothes *et cetera*.

The mistakes to be avoided in using glue are:

1. Working in the presence of a draught of air; this is fatal, as it causes the glue on the skin to dry too rapidly.
2. The glue being too hot, too cold, too thick or too frothy.
3. Taking too much glue on the brush.
4. Rubbing too thick or too extensive a layer of glue into the skin.
5. The brush being allowed to accumulate a mess of cooled thick tacky glue.
6. Exerting traction before the glue is properly set. At least half an hour should be allowed to elapse before anything greater than a slight steadying pull is used.

It may seem that this technique is described in unnecessary detail, but attention to detail is essential in applying glue. Its application is a skilled act learnt only by experience, and the description given is the result of much trial and error, and is calculated to give uniformly good results.

The Sinclair Footpiece.

In regard to Sinclair's footpiece, applying the glue too thickly, and leaving the edges of the glued strips exposed to accidental trauma, tend to result in "flaking-off" of the glued strips in one sheet. In addition, the utilization of the flannel strips not only as extending agents, but also as a support for the foot, results in an oblique shearing force being exerted at their attachment to the skin surfaces adjacent to the ring carrying the tape. Lastly, separation of the strips tends to occur at the point where they bridge the concavity between the malleoli and the heel.

To minimize this definite tendency to peeling off of the strips, the following technique has been evolved.

The Apparatus.—The apparatus required is as follows: (i) Glue, glue pot, stirrer and brush. (ii) Gauze (butter muslin) folded to form a bandage of six thicknesses, three inches wide by thirty-six inches long (see Figure I). (iii) Nine flannel strips seven-eighths to one inch wide by sixteen inches long (narrow strips tend to stretch and tear under the extending strain). (iv) Sixteen brass curtain rings of one inch external diameter bent with pliers to the shape of a triangle (see Figure II). (v) Sixteen tapes nine inches long. (vi) Small quantity of wool. (vii) Two inch strapping. (viii) One padded Sinclair's footpiece, with or without serrations. (ix) One safety pin.

The Procedure.—The gauze bandage is glued to the sole, the foregoing technique being used, so that one end extends to the heel end of the sole. It is unnecessary to carry it under the heel (see Figure I). The free upper end is interlaced amongst the toes to keep it out of the way (see Figure II). Each of all but one of the flannel strips is threaded with two curtain rings, each ring having a tape attached. Each flannel strip is then glued to the ankle and foot, the technique for glueing described above being used. The strips are distributed as follows.

The first strip is applied along the lateral side of the *tendo Achillis* as close to the mid-line posteriorly as possible. The sole is not glued, but the rings are drawn, for the time being, to the centre of the sole (see Figure II). The remaining

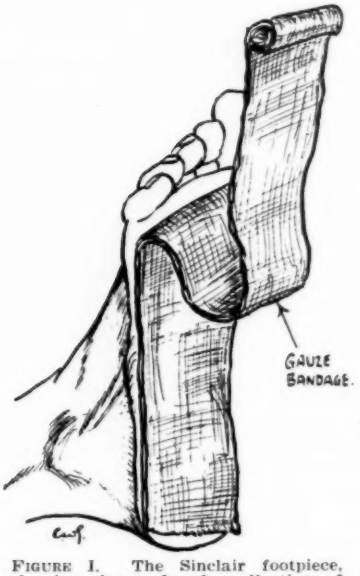

FIGURE I. The Sinclair footpiece, showing the mode of application of the gauze bandage support for the foot.

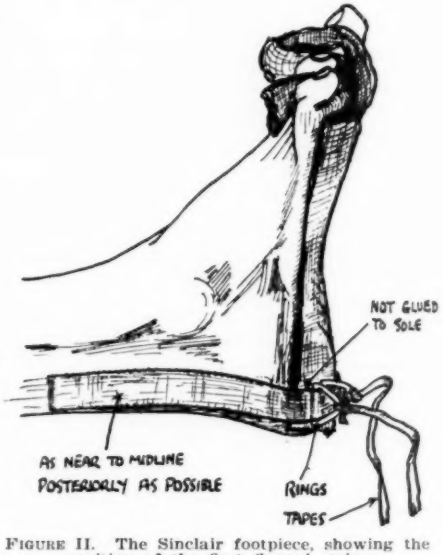

FIGURE II. The Sinclair footpiece, showing the position of the first flannel strip.

seven strips with rings and tapes are then applied similarly, from below upwards, in such a manner that they overlap their predecessor by about one-eighth of an inch, each time glueing the requisite amount of skin for one strip only. As many as possible are attached posterior to the malleoli, because these will transmit most of the pull when the extending force is applied. As successive strips are applied, it will be found that they tend to overlap the dorsum of the foot, in which case they should be glued to the skin only as far as to within one-half an inch of the mid-line (see Figure III). If glued right to the mid-line, they prevent any subsequent swelling of the foot and also are apt to produce pressure sores. The strips are applied until the last one is level with the metatarso-phalangeal articulation of the big toe. The later two or three strips serve not so much for extending purposes as for preventing the foot rotating laterally upon the foot-piece. The loose ends are then loosely (with emphasis upon the word "loosely") interlaced across the dorsum of the foot and their extremities are anchored into

position with a touch of glue (see Figure IV). The object of overlapping is to minimize the tendency for the edge of the strips to be accidentally peeled off the skin by bedclothes *et cetera*. The reason for overlapping loosely is to allow for any subsequent swelling of the foot. For the same reason, the proximal extremities of the strips are loosely bound with a circular band of strapping (see Figure V). Next, the remaining flannel strip is firmly glued to the superficial surface of the flannel strips already in position, in such a way that it passes vertically upwards parallel to the sole from a point just proximal to the heel to a point on the dorsum of the foot just proximal to the toes. It is so placed that it will firmly press the strips into contact with the skin in the concavity existing

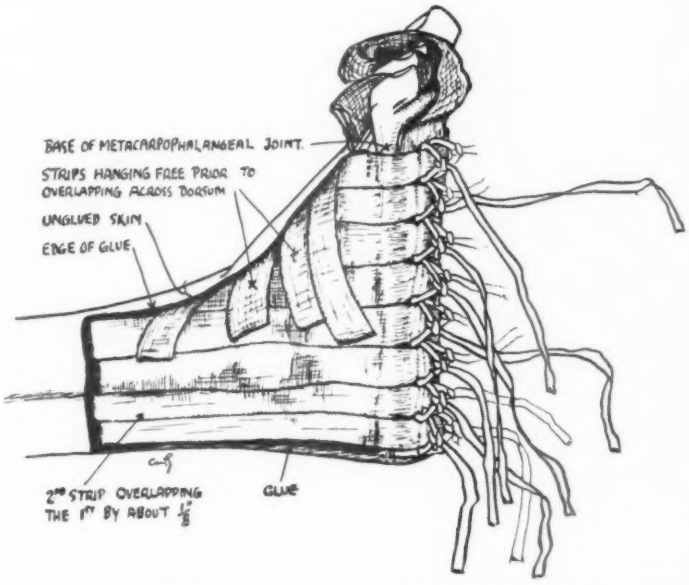
BASE OF METACARPOPHALANGEAL JOINT
STRIPS HANGING FREE PRIOR TO OVERLAPPING ACROSS DORSUM
UNGLUED SKIN
EDGE OF GLUE
2ND STRIP OVERLAPPING THE 1ST BY ABOUT 1/8"
GLUE

FIGURE III. The Sinclair footpiece, showing a further stage of the application of the flannel strips.

between the malleoli and the heel. It should be separated from the *tendo Achillis* by a small pad of wool, and its object is to overcome the great tendency to separation of the strips from the concavity mentioned (see Figure V). Finally, the rings are drawn to the lateral border of the sole.

The padded footpiece is applied as follows: The limb is placed in the splint upon the flannel slings and the foot is held up by means of the gauze bandage glued to the sole. The foot piece, with the horizontal bar adjusted at the correct level (the commonest mistake is to have the bar too high), is then placed against the sole with the horizontal bar resting upon the side bars of the splint. The foot is then elevated until the malleoli are at the correct level, that is, level with or just below the horizontal bar, so that the application of the extending force results in the foot assuming a position at right angles to the leg or of slight foot drop. The gauze bandage is then drawn taut over the top and down the back of the footpiece, turned around latter's thumb-screw and drawn up to the top edge

of the footpiece, where it is attached by a safety pin running transversely (see Figure VI). The tapes attached to the rings are then tied around the back of the footpiece in the usual manner, preferably after threading one tape of each pair between the gauze bandage and the footpiece. Extension is then applied to the horizontal bar.

It is now seen that the weight of the foot is taken entirely by the gauze bandage (which also allows the height of the foot to be adjusted from time to time), that the lower flannel strips are used only for extending purposes, and that the upper flannel strips are used partly for extending purposes and partly for preventing lateral movement of the foot on the footpiece (see Figure VI). That is, the shearing-off action, present when the strips are used for supporting the weight of the foot as well as for extension, is eliminated. Also, the use of the gauze bandage for supporting the foot obviates the necessity for serrations along the sides of the footpiece. The writer uses a footpiece consisting of a rectangular piece of wood three and one-eighth by eleven inches by half an inch, with no serrations (see Figure VI).

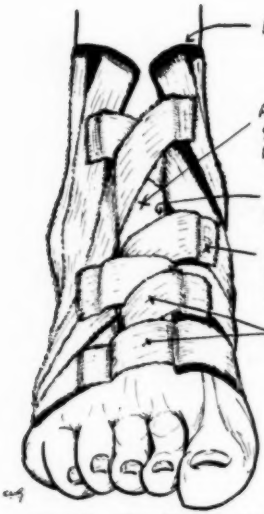
EDGE OF GLUE.
A STRIP 1" WIDE OF SKIN OF DORSUM NOT GLUED.
MEDIAL LIMIT OF GLUE ANTERIORLY
END ANCHORED WITH A TOUCH OF GLUE.
OVERLAPPED LOOSELY

FIGURE IV. The Sinclair footpiece, showing the technique of overlapping the flannel strips across the dorsum of the foot.

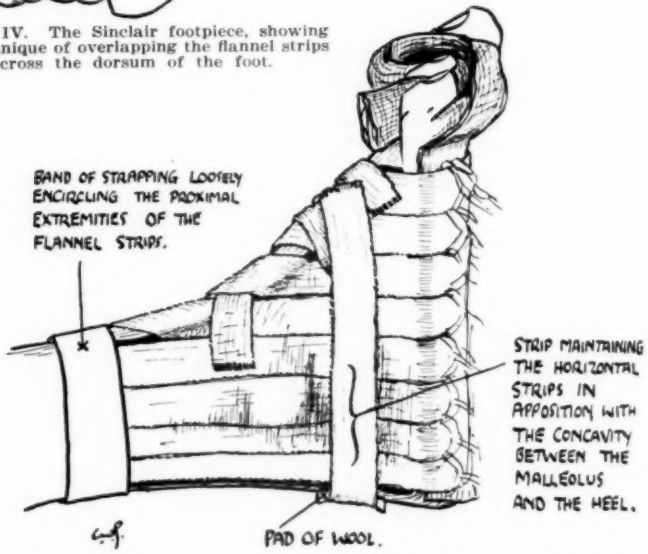
BAND OF STRAPPING LOOSELY ENCIRCLING THE PROXIMAL EXTREMITIES OF THE FLANNEL STRIPS.
STRIP MAINTAINING THE HORIZONTAL STRIPS IN APPOSITION WITH THE CONCAVITY BETWEEN THE MALLEOLUS AND THE HEEL.
PAD OF WOOL.

FIGURE V. The Sinclair footpiece, showing the application of the strapping and the last strip of flannel.

Application of the footpiece by this technique will reduce to a minimum any tendency for the strips to become detached.

The Mechanics of Extension Systems.

Extension systems possess several components of which the first is always kept in mind, but the remainder are often partly or seemingly completely forgotten. They are: (i) Extension forces, (ii) counter-extension forces, (iii) angulation correction forces, (iv) rotation correction forces, (v) support for the limb. The fifth is often an innocent ill-doer in that it nullifies the extending forces while not in any way acting as a counter-extending agent.

In the writer's opinion, students should not be taught that such-and-such a fracture may be treated by A's method, or Mr. X-Y's method, or with B's splint. Rather, he should be instructed (assuming that he is already aware of the principles governing the treatment of fractures) as to the various methods of applying an extending force, of applying counter-extension and of supporting the limb *et cetera*. He should be taught the most advantageous ways of combining these forces, and how to decide which combination is most suited to the particular fracture he is called upon to treat. Thus he would be taught that the available means of extension are "mobile" (that is, by a cord passing from the distal fragment through a pulley to a weight), "fixed" (the distal fragment being anchored to the end of a splint by a non-elastic agent, such as gauze bandage or cord), and "elastic" (see Figure VI); that the available means of counter-extension are the weight of the patient's body, the attachment of the upper end of a splint to the limb by strapping or glue *et cetera*, and the pressure of the splint against a fixed bony point, for example, the ischial protuberance, the lateral chest wall *et cetera*.

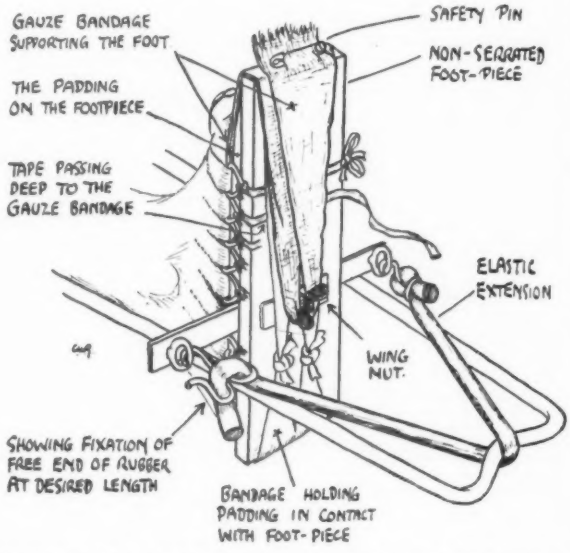
GAUZE BANDAGE SUPPORTING THE FOOT
SAFETY PIN
NON-SERRATED FOOT-PIECE
THE PADDING ON THE FOOTPIECE
TAPE PASSING DEEP TO THE GAUZE BANDAGE
ELASTIC EXTENSION
WING NUT
SHOWING FIXATION OF FREE END OF RUBBER AT DESIRED LENGTH
BANDAGE HOLDING PADDING IN CONTACT WITH FOOT-PIECE

FIGURE VI. The Sinclair footpiece, showing (1) the non-serrated footpiece, (2) the method of fixing the gauze bandage, (3) the method of tying the flannel strips, (4) the method of applying the elastic extension.

He would be taught that the available means of correcting angulation are continuous elastic pull, mobile traction on an encircling band, and direct pressure against the angulation; that undesirable rotation may be prevented by adjusting the cords supporting the splint, and so on.

He would be taught that mobile extension by weight over a pulley, the limb being supported by slinging it in a Thomas or similar splint, and the weight

of the body being used as counter-extension, makes a good combination for a fractured femur.

He would be taught that mobile extension and counter-extension against the ischial protuberance are mechanical incompatibles.

Finally, he should be urged that, after completing the erection of any and every extension apparatus, he should stand back, survey the finished job, and repeat to himself the following catechism: "What factors have I exerting an extending force in this case?" "What factors have I exerting a counter-extending force?" "Do the combinations I have chosen work harmoniously together?" "Have I guarded against any undesirable rotation or angulation, and, if, not, how can I best do it?" "Do any of my various supports, angulation prevention forces *et cetera*, tend to nullify my extending forces?" And finally: "Is my device actually extending the limb?" To these it occasionally happens that the answer, to all practical purposes, is in the negative.

Let us now consider these five components of an extension system in order, and discuss the common mistakes occurring in their application.

Extension Forces.

Fixed Extensions.—The term "fixed" extension is applied to those extension systems in which the proximal part of the splint is fixed to the limb or body proximal to the fracture, whilst the distal fragment is drawn upon and fixed to the distal end of the splint.

The agents utilized for attaching the limb to the distal end of the splint fall into one of two groups. They are either (i) non-elastic, such as cord or gauze bandage, or (ii) elastic, such as rubber. There is considerable difference of opinion as to which is theoretically the more correct.

Concerning non-elastic agents, reflection will show that an extending force is being exerted upon a limb placed in this form of "extension", only for the few seconds or minutes that the surgeon is actually pulling on the limb immediately prior to knotting the "extending" agent to the end of the splint. Immediately the knot is tied, the system becomes one in which two opposing forces are merely maintaining the limb in the degree of extension in which the surgeon has placed it.

Supposing that the agents exerting these forces (that is, the cords, gauze bandages *et cetera*) are absolutely incapable of stretching, then theoretically they will maintain the limb in the position in which it has been placed for an indefinite time. Actually what happens is this. The patient remains quiescent for a while until the pain or discomfort resulting from the surgeon's manipulations has subsided; then he finds he has slipped down in the bed or is in some other irksome position; he pulls himself up or moves his body in some other manner and unconsciously contracts the muscles of the injured limb, so exerting a force upon the "extending" agents. The cord (or bandages *et cetera*) is inextensible; therefore the knots tighten. He relaxes himself and, the knots having tightened, the cord is now longer; consequently the limb now lies without fixation and is able to become shorter. Or else his foot, encumbered by a footpiece, gives him the sensation of being stiff or otherwise uncomfortable, and so he "waggles" it and so exerts a temporary extra strain on the knots, with the same result.

Time after time, examination of such an extension a few hours after the surgeon's tightening-up process, reveals loose cords.

No person can tie a knot in a cord that is incapable of further tightening. If a cord is stretched to its capacity by ordinary manual means between two immovable objects and anchored by knots tied as tightly as possible, a few hard pulls transverse to the cord will further tighten the knots and allow the cord to sag.

Even if the cords are tightened six times a day, the maintenance of the extension produced by the surgeon is not continuous. Quite apart from this consideration, intermittent extension is wrong in principle.

So it appears that in every twenty-four hours a patient fitted with fixed extension with a non-elastic extending agent, experiences a few minutes' forcible

extension, followed by a varying period up to a few hours of immobilization and maintenance of this degree of extension. This in turn is followed, for the remainder of the twenty-four hours, by the limb being allowed to lie unextended and unimmobilized in the slings of the splint.

If, however, we replace the non-elastic cord with an elastic extending agent, such as a round rubber length, then the picture becomes entirely different. When the leg is put up originally, the elastic strain is adjusted to whatever degree the surgeon thinks necessary. From then on, the extension requires no more than regular supervision, is quite continuous, is perfectly comfortable, and the degree of pull steadily diminishes as the lower fragment passes distally to its normal position. So that, when full extension is obtained, the extending force is often diminished to that just sufficient to maintain the extension. In any case, it is readily adjustable to the needs of the case. Furthermore, if a temporary extra strain is thrown upon it by the patient's movements, it gives to the strain, but takes up its original extending force immediately the patient again becomes quiescent.

The writer uses an extending agent which can be manufactured for a few shillings, consisting of one piece of solid round tourniquet rubber of seven-sixteenths of an inch diameter and twenty inches long. (This diameter has been found to be the most useful after much trial.) Its construction is shown in Figure VII, the hooks consisting of soft iron wire bent with pliers to the requisite shape. The free end of the rubber is turned around the free hook at the length required (see Figure VI). An inverting or everting pull can be exerted if necessary.

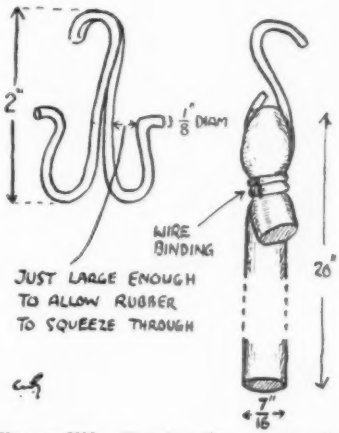

FIGURE VII. Showing the components of the rubber extension illustrated in Figure VI.

Counter-Extension Forces.

Common errors in the application of these forces are as follows:

1. Where pressure of the ring of a Thomas splint against the ischial protuberance is used as counter-extension, unless the ring of the Thomas splint is specially fitted to the case being treated, the writer and most house surgeons experience difficulty in maintaining it in apposition with the protuberance. A pull on the splint upwards and inclined towards the head of the bed helps to maintain it in position, while utilization of a flannel band passing from side bar to side bar under the thigh to correct angulation tends to pull the ring downwards away from the protuberance. Occasionally, when the ring does slide off the protuberance and rides against the pubis, it is well tolerated and is efficient. Argument in its favour is described by Sinclair,[2] argument to the contrary by Corlette.[6] The writer's experience fits in with Corlette's remarks.

2. Where the proximal end of the splint is attached to the thigh. Glued gauze bandages are preferable to strapping. A common mistake in using strapping is to apply the Thomas splint with the ring not far enough distal to the hip. The usual slipping of the strapping occurs, and eventually the ring approaches the pelvis and produces discomfort, and, in the case of the tibia splint, the amount of distal end of the splint available for application of the fixed extending force becomes less and less and finally insufficient.

3. Where the patient's body weight constitutes the counter-extending force, the essential raising of the foot of the bed on blocks is often insufficient and occasionally entirely omitted.

Angulation Correction Forces.

In the treatment of a fractured shaft of a femur, in the Hamilton Russell extension a soft pillow should be utilized to support the site of fracture, but is very frequently forgotten. For this reason Mr. Russell has expressed his regret that he did not stress its importance sufficiently in his article describing the application of this extension.[7]

When counter-extension is used against the ischial protuberance, a tight flannel bandage, passing from side bar to side bar of the Thomas splint and under the thigh, is often applied. This has the disadvantage of tending to push the ring backwards away from the protuberance. A more satisfactory method, not having this disadvantage, is to pass a broad sling under the site of angulation, and attach it to a cord passing in the direction desired to a pulley over which it passes to a suitable weight.

Irrespective of the fracture, the pull (or push) must be continuous.

Rotation Correction Forces.

Rotation correction forces may be discussed in connexion with the supports of the limb.

The Supports for the Limb.

The supports of the limb often have a most injurious effect upon the preceding four forces. The discussion will be confined to fractures of the tibia and femur in which the limb lies in a sling passing under it from side bar to side bar of a Thomas or similar splint, the splint *plus* limb being supported by cords passing from the splint to an overhead frame. Consider (i) the supporting cords and (ii) the sling.

The Supporting Cords.—A quite common mistake in treating a fractured femur by slinging it in a bent Thomas knee splint with the foot attached to the end of the splint, with the end of the splint drawn on by a weight over a pulley at the end of the bed, and with the weight of the patient's body being utilized as a counter-extension, is to have one or all of the supporting cords of the splint passing in a direction obliquely upwards and towards the head of the bed. These supports are obviously nullifying the extension force without contributing to counter-extension. The reason that the supports are directed towards the head of the bed is the, at first sight, natural one of pulling the splint up against the posterior aspect of the femur so that a flannel band passing from side bar to side bar underneath the thigh can be utilized to produce a slight anterior bowing of the femur or to correct a posterior bowing. The remedy for this is not to utilize the flannel bands passing between the side bars, but to pass a sling under the site of angulation and to attach a cord which is passed to a weight over a pulley in the direction desired.

All cords supporting splints should pass either directly vertically, or obliquely upwards and towards the foot end of the bed, when the weight of the body is used as a counter-extension.

When the counter-extension is produced by pressure against the ischial protuberance, the splint-supporting cords should be inclined towards the head of the bed so as to help to maintain the splint in contact with the protuberance. Even in these circumstances the flannel band passing from side bar to side bar under the thigh should not be used to correct angulation, because the more it is tightened, the more it tends to move the ring of the splint downwards and backwards from the ischium.

The writer has often received the reply when he has pointed out the extension nullifying action of a supporting cord: "Oh, that little force won't affect the extension much; the weight is too little." This statement is entirely wrong, because all available factors should be aiding either extension or counter-extension.

Another common mistake in the method used for supporting a splint is as follows. Often supporting cords are placed at each end, each support consisting of a cord, tied to one side bar of the splint and passed up to and through a loop

in a supporting cord and downwards to be tied to the opposite side bar. Passing the cord through a loop in this manner allows the splint to rotate in any manner.

A more satisfactory way (see Figure VIII) is to tie one end of one cord to one side bar and the other end to a ring suspended from a single supporting cord, and to have a second cord passing from the ring to the opposite side bar fitted with a wooden toggle as described by Corlette[6] (see Figure VIII). The toggle, which consists of a piece of wood three inches by one inch by half an inch, with a hole bored at each end of a diameter just larger than the cord used (see Figure VIII), allows of the length of this cord (and consequently the rotation of the splint) to be adjusted rapidly at will. Within limits, the thicker the toggle and the nearer the diameter of the holes approach that of the cord, the more efficient it is.

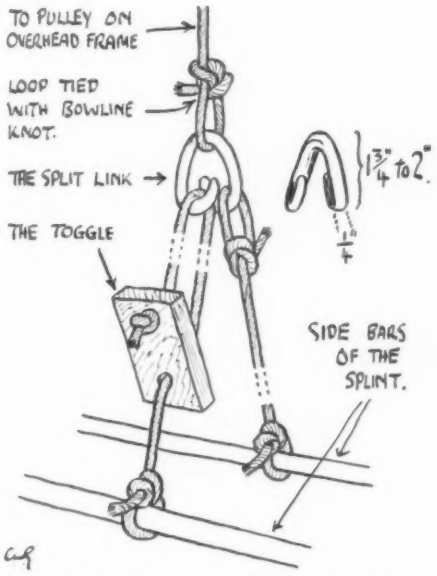

FIGURE VIII. Showing (1) the split link, (2) the toggle, (3) the method of slinging a splint.

Another useful instrument is the split link (see Figure VIII). These are useful links for connecting each pair of splint supporting cords to its corresponding upper single one. They are also useful as hooks for attaching the weights to the end of the cords. They obviate much time wasted in untying knots. They are obtainable by the dozen at any large ironmongery, the most useful being one and three-quarter to two inches long by one-quarter inch diameter.

The Sling.—Various types of sling are described by Corlette[6] and others, of which the commonest is a series of flannel bandages looped around one side bar and attached to the opposite by paper-clips (which tend to allow the bandage slings to slip) or safety pins.

These slings may, in addition to acting merely as slings, act as extension nullifying, counter-extension aiding or angulation allowing forces.

These secondary actions will be illustrated by a discussion of each of the extending systems illustrated in Figure IX. The fractures discussed and the method of extension and counter-extension utilized in each case, are described in the figure.

In case "a", for the limb distal to the fracture to pass distally under the strain of the extending agent, it must either slide over or drag with it all that portion of the sling it is resting on, distal to the fracture.

In addition to this extension nullifying friction factor, owing to the line of traction being horizontal, the portion of the sling extending from the popliteal space to the site of fracture will press against the under-surface of the thigh and tend to prevent it passing distally. The greater the degree of flexion of the knee, the greater the degree of this latter force.

The remedy is to avoid excessive flexion of the knee and to insure that the sling between the popliteal space and site of fracture is loose, or to use another method of extension.

In case "b", where the splint is theoretically a support and nothing else, the degree to which the foregoing factors operate depends upon the direction of the supporting cords. The more the latter are inclined towards the head of the bed, the more the foregoing factors operate. If the cords are inclined slightly towards the foot of the bed, the splint is allowed to move distally with the limb distal to the fracture. This system is not so efficient as system "c" because the

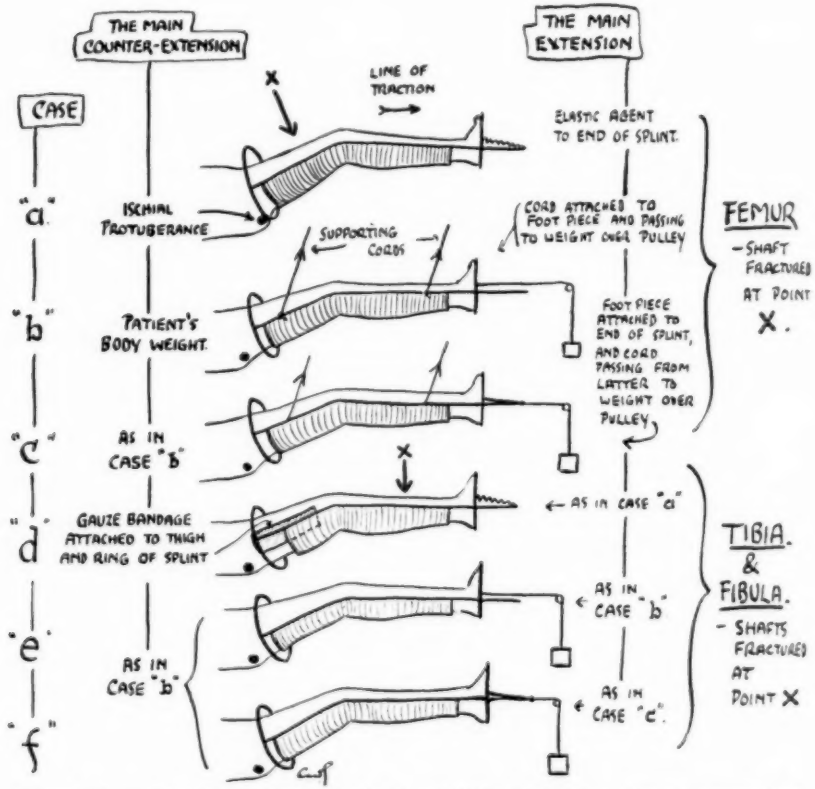
THE MAIN COUNTER-EXTENSION
THE MAIN EXTENSION
CASE
LINE OF TRACTION
ELASTIC AGENT TO END OF SPLINT.
"a"
ISCHIAL TUBERANCE
CORD ATTACHED TO FOOT PIECE AND PASSING TO WEIGHT OVER PULLEY
FEMUR - SHAFT FRACTURED AT POINT X.
SUPPORTING CORDS
"b"
PATIENT'S BODY WEIGHT.
FOOT PIECE ATTACHED TO END OF SPLINT, AND CORD PASSING FROM LATTER TO WEIGHT OVER PULLEY
"c"
AS IN CASE "b"
"d"
GAUZE BANDAGE ATTACHED TO THIGH AND RING OF SPLINT
← AS IN CASE "a"
TIBIA & FIBULA. - SHAFTS FRACTURED AT POINT X
"e"
AS IN CASE "b"
← AS IN CASE "b"
"f"
← AS IN CASE "c".

FIGURE IX. Illustrating various methods of combining extension and counter-extensions, and various secondary actions of the support for the limb.

pull of the supporting cords is not utilized to augment the main extension, and because the control of the limb, owing to its lack of fixity to the splint, is not so positive.

In case "c" the limb distal to the fracture is fixed by an inelastic, inextensible agent to the end of the splint. Consequently it lies continuously in a fixed relationship to the splint, except for that portion proximal to the knee. Reference to the diagram will show that, owing to the extending force passing parallel to the leg distal to the knee, the portion of the splint proximal to the knee

bend will pass horizontally distally as the lower fragment of the fracture passes down to its normal position. As a result, the sling between the popliteal space and the site of fracture falls away from the under-surface of the thigh and so does not combat any tendency to gravity angulation. In this case it is imperative that the supporting cords of the splint do pass with a distal inclination, and in these circumstances, in distinction to case "b", they do augment the extending force. This is the method of extension the writer prefers for this type of fracture.

Now consider the same types of extension used with fracture of the shafts of the tibia and fibula.

In case "d", as in case "a", we have the same considerations as to friction operating with that portion of the sling distal to the fracture, but, the area being so short, this factor is practically negligible. In this case, however, the friction factor of the sling proximal to the fracture is an advantage in that it aids counter-extension. If it happens that a Thomas knee splint is being used, then a considerable degree of counter-extension is found to be exerted, provided the knee is well bent, by the pressure of the sling proximal to the popliteal fossa against the posterior surface of the thigh.

In case "e" the friction factor is negligible, but the system has the disadvantage of comparative lack of control of fixation and rotation of the lower fragment.

In case "f" the considerations are the same as for case "c". For fractured tibiæ requiring extension, the writer prefers the system "d", utilizing a Thomas tibia splint with rocker, and with counter-extension by fixation of the thigh to the ring of the splint by gauze bandage glued to the thigh, and with an elastic extension to the end of the splint *via* a Sinclair footpiece.

Summary.

1. The commoner mistakes in treating fractures, observed during two and a half years' experience as house surgeon, are described, with suggestions as to their avoidance and correction.

2. The advantages and disadvantages of glue and adhesive strapping as means of applying an extension are discussed, together with hints as to how to overcome the disadvantages of each.

3. An efficient technique, with the necessary apparatus, for glueing materials to the skin surface is described.

4. An account is given of an efficient technique for applying the Sinclair footpiece, with a description of a simplification of it.

5. The various components of an extension system are discussed in order.

6. A scheme for the tuition of students in the principles of extension *et cetera*, is suggested, with emphasis upon the importance of finally surveying every extension apparatus immediately after its application is completed.

7. The term "fixed extension" is analysed, non-elastic fixed extension being criticized adversely, and elastic extension favourably. The construction of a simple elastic extending agent is described.

8. The methods used for supporting splints are discussed, common mistakes being described, with hints as to their correction.

9. The existence of various, often unsuspected, secondary actions of splint and limb supporting agents in various commonly used methods of extension is disclosed and discussed.

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(1) C. E. Corlette: "The Treatment of Fractures, with Special Reference to Those of the Lower Limb", THE JOURNAL OF THE COLLEGE OF SURGEONS OF AUSTRALASIA, March, 1930, page 349.

[2] Meurice Sinclair: "The Use of the Thomas Splint in Fracture of the Long Bones", THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY, September, 1931, page 156.

[3] Meurice Sinclair: "The Thomas Splint", 1927, pages 25, 160, 162.

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[5] Meurice Sinclair: "The Thomas Splint", 1927, page 25.

[6] C. E. Corlette: "The Treatment of Fractures, with Special Reference to Those of the Lower Limb", THE JOURNAL OF THE COLLEGE OF SURGEONS OF AUSTRALASIA, March, 1930, page 336.

[7] R. Hamilton Russell: "Papers and Addresses in Surgery", 1923, page 278.

TECHNIQUE FOR INDUCTION OF LOCAL ANALGESIA IN THE LOWER URINARY TRACT BY PARASACRAL INJECTION.

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Premedication.

As has been suggested by Dr. C. E. Corlette, a preliminary injection of a quarter of a grain of morphine with one-hundred and fiftieth of a grain of hyoscine given one hour before operation, and supplemented, if necessary, by another one-sixth of a grain one hour later, banishes the fear of operation, diminishes the discomfort from the lithotomy position in which the patient must be placed for the parasacral injection, and tends to make the patient sleep during the operation. The disadvantages of this measure are that the patient complains of dryness of the mouth and often groans throughout the operation. The mouth dryness is easily relieved by saturating a gauze swab with water and allowing it to drop thence to the side of the mouth. The groaning is disconcerting and at first may be interpreted as failure of anæsthesia, but in several of our cases the groaning continued before the patient was touched, and subsequent close questioning of the patients revealed that they remembered only the pain of inserting the needles.

Solution Used.

In all cases a 0·5% solution of "Novocain" with one in 150,000 to one in 200,000 adrenalin in normal saline solution was used. It is essential that either the solution be freshly prepared or else the adrenalin be added to a previously made solution of "Novocain" immediately before use. The "Novocain" solution without adrenalin and in the absence of compounds of sodium can be boiled, but adrenalin is destroyed by heat. When adrenalin has been added, the solution should be either colourless, becoming pinkish, or a light pink from the first. Any tendency to a brownish colour should arouse suspicion of oxidation and the solution should be discarded. About 300 cubic centimetres (ten fluid ounces) of this solution are required for the parasacral block, together with the abdominal field block to be described later.

Although "Novocain" is definitely a poison in large doses, the addition of adrenalin so greatly retards its absorption that up to 480 cubic centimetres (16 fluid ounces) of the 0·5% solution of "Novocain" with adrenalin can be injected without any apparent toxic effect.

Armamentarium.

A 10 cubic centimetre to 20 cubic centimetre glass syringe with a device for locking the needle is required. The Labat type of lock chiefly is favoured because of the superiority of needles of French manufacture. At present a glass 20 cubic centimetre syringe with an eccentric nozzle is used, the Labat locking device being added by Elliott Brothers. (Rings on the cap of the syringe are advantageous.) A fine hypodermic needle is required to raise wheals for the puncture with the bigger bore needles. The needle used for the deep injection is at least 12·5 centimetres in length and, if possible, one or two centimetres longer, as it

is unwise to insert the needle right up to the mount, since this is the weakest point of the needle and the site of likely fracture.

A pair of ordinary dissection forceps are necessary to grasp the needle towards the point so as to facilitate the puncture through the skin, toughened considerably by the intradermic injection.

The Parasacral Injection.

If the sacrum is looked at in profile, it will be seen that from the sacro-coccygeal junction to the second sacral foramen is practically a straight line. On this fact depends the possibility of blocking the whole ten sacral nerves as they emerge from the anterior foramina.

To prepare for the injection, the tip of the coccyx is found, two points from one and a half to two centimetres are taken on either side of this bone, and a wheal is raised by intradermic injection. Taking the long needle by the mount unattached to the syringe in one hand, and the needle towards the point grasped by forceps in the other hand, the needle is inserted through the wheal.

The lower end of the sacrum is then searched for and, when found, the needle is guided anteriorly over it, then pushed straight on till bone is felt. If no blood issues from the needle, a few drops of the solution are injected and the syringe detached again. If the solution is not blood-stained, the injection is continued. If it is blood-stained, the needle is withdrawn and reinserted in a slightly different position until no blood is seen. Then the syringe is attached and injection slowly made whilst withdrawing, pausing at times, but always injecting until the needle is again subcutaneous, using for this injection from 30 to 40 cubic centimetres. When the needle is subcutaneous the needle is reinserted, the hand holding the mount being slightly depressed, thus aiming for the bone above the first foramen. When bone is felt, 40 cubic centimetres are injected with the needle still in position, the above-mentioned precautions regarding blood being again observed. The opposite side is similarly injected. Quite often here one will tap the previously injected fluid which has found its way across the mid-line behind the rectum.

Too much stress cannot be laid on the dangers of injecting the solution directly into the vascular system, but if the precautions above are taken, no bad effects will ensue.

Attempting to suck blood back by using the syringe is not a reliable method of excluding puncture of a vessel, since by the suction tissue may be drawn into the lumen of the needle and no blood may appear.

When the deep injection is completed our attention is directed to the coccyx. From the two previously made wheals about 10 cubic centimetres are injected in front and 10 cubic centimetres behind the coccyx to block the coccygeal nerves.

Then after having injected subcutaneously the line joining the two "coccygeal" wheals the long needles are inserted through the punctures and guided anteriorly towards the pubic bone. The needle must be kept in the subcutaneous plane and a subcutaneous strip injected with 10 cubic centimetres of solution whilst withdrawing the needle.

In suprapubic prostatectomy these last-mentioned subcutaneous injections are not necessary, but if the perineum is to be made completely insensitive, as for perineal prostatectomy, they are essential.

The Abdominal Field Block.

By means of the small hypodermic needles the following punctures are made to raise wheals: one on each side level with the umbilicus and at the outer border of the recti muscles, two in the corresponding position above the pubis, one over the *symphysis pubis*, one just below the umbilicus. From the lateral punctures the long needle is inserted into the rectus sheath. The needle is then passed transversely through the deep fascia above the pubis and below the umbilicus and deep injections are made. The needle is then inserted through the wheal above the symphysis, the pubic bone felt for and the needle passed

behind it. Ten cubic centimetres are then injected into the cave of Retzius. The needles are then inserted through the wheals and the subcutaneous tissues injected round the whole field. Lastly, from the wheal below the umbilicus a median subcutaneous strip is injected on the line of the incision. The actual line of mid-line incision is thus left free from distortion by local anæsthesia.

By the time the abdominal injection is completed the field is usually ready for operation. If only the perineum is the site of operation, it is wise to wait five or ten minutes to insure complete anæsthesia. The nervous tissue has not been directly injected, so that time is required for dispersion of the anæsthetic solution. This delay before commencing operation is most important, and omission of it is one of the causes of apparent failure.

The relaxation of the muscular tissue is most striking. The paralysis of the *sphincter ani* is particularly noticeable, allowing very thorough examination of the prostate and bladder floor. The tissues in the field of operation become almost bloodless from the action of the weak adrenalin solution. This fact is at first disconcerting in that the bladder, owing to its marked pallor, is not so easily recognized. The adrenalin also appears to have an effect on the blood supply of the prostatic cavity, as the arterial bleeding from the torn edge of the bladder mucosa overhanging the prostatic bed and the hæmorrhage from the prostatic bed itself in most cases have been greatly diminished. This anæmia, too, lasts apparently long enough for the vessels to be occluded by blood clot, for up to now no reactionary hæmorrhage has occurred.

Sometimes difficulty has been encountered in reaching the deep urethra in prostatectomy; although pain has been abolished, the patient can feel movement. This causes him to contract the unparalysed muscles. This defect can, however, be easily overcome by gently exerting steady pressure with the fist of the enucleating hand, when the tissues will be felt to yield.

In our first attempts, failures to produce anæsthesia were not uncommon. Most of these were due to three causes, namely, (i) fear of puncturing important structures with the needle, (ii) fear of injecting too much "Novocain" solution, and, lastly, (iii) attempts to commence operation before sufficient time had elapsed after injection.

Nevertheless, after eliminating the above causes, we have had failures which are as inexplicable to us as those found at times with spinal anæsthesia, but on the whole the success of this method has been so marked as to make us prefer it as the method of choice for operations on the lower urinary tract.

OPERATIONS OF THE URINARY TRACT UNDER LOCAL ANALGESIA, WITH PARTICULAR REFERENCE TO PROSTATECTOMY.

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Sydney Hospital.

Between what matters and what seems to matter,
How should the world we know judge wisely?—E. C. Bentley.

Chloroform and ether have had a triumphant reign as anæsthetics, but in recent times from many critical surgeons have been heard the mutterings of revolt, and open insurrection has broken out in many places.

A surgical operation is a severe ordeal for anybody, and there is no doubt that many die from operations done under general anæsthesia who would survive if anæsthetic toxæmia and shock were eliminated.

Anyone who has done much general surgery and reflected on it, cannot fail to think that surgery would be better and safer if general anæsthetics could be abolished and if painful and other stimuli could be abolished also. The medical literature is now full of the results of ardent research workers in this direction. In *The Medical Journal of Australia*, February 7, 1931, there is an excellent article by C. E. Corlette, in which he summarizes the whole matter in a masterly fashion, and in addition gives his own extensive experience.

It is not my intention in this paper to attempt a comparative study of the various methods of producing local analgesia. Let me say that, as far as my experience goes, I am with Corlette in everything he says. I know that medical men are apt to turn very readily to strange and new gods. The amused sceptic smiles and bides his time, knowing full well that the miracle of today will be forgotten tomorrow. But it is also a great misfortune in this life to suffer from what Bernard Shaw calls the "time lag", that is, a peculiar obtuseness which will not allow one to see a new thing and a good thing when it is literally staring one in the face.

In urological surgery the scope for local analgesia is for many reasons very great and it is extensively practised. The local application of a sufficient concentration of a suitable anæsthetic to the urethral and bladder mucosa will for the most part be quite suitable for numerous cystoscopic examinations and minor or even major surgical procedures. I have on a number of occasions used fulguration for papillomata of the bladder and performed litholapaxy with no other anæsthesia. But for very sensitive patients, or for those with very painful lesions of the bladder, something more becomes necessary.

Apart from any other objection, general anæsthesia has the disadvantage that the patient is unconscious at a time when it may be most important that he be conscious. For example, I consider it very unwise to perform pyelography in an unconscious patient. In such cases resort to some form of local analgesia is necessary.

Spinal analgesia has had an extensive trial, and the literature on this subject alone is enormous. It was first employed by Bier in 1899. Cocaine was used, and many deaths resulted. After the discovery of "Novocain" in 1904 by Einhorn, comparative safety was reached. Since then many modifications of technique have been introduced from time to time, for the very obvious reason that unpleasant

and dangerous complications arose. Even with the most modern technique these have not been completely eliminated.

I have given spinal analgesia an extensive trial, my first experience of it being in 1912, and I used it extensively during the war. Many writers whose experience is vast, are most enthusiastic about it, and we cannot deny that spinal analgesia in selected cases is a most valuable adjunct to our methods of producing local analgesia. However, I am sure that from a point of view of absolute safety it is vastly inferior to other methods which also have had an extensive trial.

Caudal analgesia was first suggested by Cathelin in 1901, and first used on a human being by Stoekel in 1909. Alone or together with transsacral block, it has had a tremendous vogue in urological clinics in America and elsewhere. It is extensively used at the Mayo Clinic. For those who are interested, there is a complete and voluminous literature available.

Owing to certain technical difficulties and a percentage of unexplained failures and complications, the method has its limitations. Nevertheless, we have here a distinct advance over spinal analgesia. I have given caudal analgesia a good trial and can speak very highly of it.

It is the main purpose of this paper, however, to present a report on my experience with parasacral analgesia alone or combined with abdominal field block for the major operations of urological surgery. In reviewing the literature on this subject, it is strange that the advantage of parasacral analgesia over caudal or transsacral block seems to have escaped the notice of most operators.

Dr. C. E. Corlette pointed out to me some time ago the advantages of parasacral analgesia over other forms, and with the enthusiastic support of Dr. Walter Perry, my Clinical Assistant at the Department of Urology at Sydney Hospital, I have now produced parasacral analgesia well over one hundred times.

It would be wise just here briefly to define a few terms. Caudal or epidural analgesia consists in introducing an anæsthetic solution through the sacrococcygeal membrane into the cavity of the sacrum. Transsacral analgesia consists in anæsthetizing the sacral nerves by puncturing the posterior sacral foramina, while parasacral analgesia consists in anæsthetizing the sacral nerves as they emerge from the anterior sacral foramina.

From our experience we have come to the conclusion that parasacral analgesia is the most satisfactory method of anæsthetizing the sacral nerves. The technique is simple and easily learned by house surgeons. The percentage of failures is low and the reactions nil.

At first we had, as we would expect, some failures, but with the standard technique now elaborated our failures are practically nil. There were no vasomotor or toxic reactions such as one so frequently sees in spinal and caudal analgesia, some of them very alarming. I must make an exception for one patient who developed an attack of abdominal pain at the end of a cystoscopic session lasting about an hour. The pain was subumbilical and gradually increased in intensity until he presented the clinical picture of a perforative lesion of the abdomen, with severe pain, sweating and a board-like rigidity. Fortunately, I was confident of every step in the examination, and so treated the patient on expectant lines. He was better in a few hours, and quite well next day. I cannot explain this peculiar reaction, but I put it down, perhaps unfairly, to the parasacral analgesia. We have had no other reactions whatever.

Parasacral analgesia is now a routine procedure in my practice for all difficult cystoscopic examinations and operations on the bladder or prostate. I shall leave a detailed description of the technique to Dr. Walter Perry, with whose cooperation this work was carried out.

A review of our cases brings to light some interesting facts which I purpose to set down. There were twenty cases of enlarged prostate gland. I do not wish to digress into a dissertation on prostatic enlargement and its operative treatment, but it must be remembered that prostatectomy is a formidable ordeal for the patient, and except when performed by an expert, and with proper ante-operative care, carries a high mortality rate.

The evolution of this operation makes a fascinating study, and in a general way it may be divided into three stages. At first the mortality from suppression of urine and uræmia was terrific, until it was realized that operation could not be attempted until the patient had undergone a sufficient period of decompression in order to restore to the kidneys their former function. In many cases this takes weeks of treatment. This principle is now universally recognized, and deaths from uræmia in urological clinics are now practically abolished. The next stage was the elaboration of an open operation in place of blind enucleation as first practised by Freyer. The modern operation consists in a visual inspection of the prostatic bed after the gland is removed. All tags of tissue and obstructive bars are removed, hæmostasis is made perfect, and adequate drainage established. In this way that formidable and often fatal complication, post-operative hæmorrhage, is abolished, and sepsis is reduced to a minimum.

These two fundamental principles have now been practised by urologists for a number of years with a striking fall in the morbidity and mortality rates.

The final phase is the abolition of cardio-vascular and pulmonary complications by the routine use of some form of local analgesia.

In reviewing the deaths following 170 prostatectomies at the Massachusetts General Hospital between the years 1926 to 1930 inclusive, Swan and Mintz (*Journal of Urology*, July, 1931), among other conclusions, point out that the mortality was 5·8%, and "the causes of death were chiefly circulatory and none were renal".

In our cases there was a striking freedom from shock or any upset whatever, with a smooth convalescence from the moment the patients left the operation table. The patients were in no way selected, for, as I have said, it has become our routine practice to perform all prostatectomies with parasacral and abdominal block anæsthesia.

A brief summary of the case histories of a few of our patients will show that they were anything but "good surgical risks".

CASE I.—W.F., aged eighty years, was admitted to hospital on November 24, 1931. This man had chronic urinary obstruction for two years, culminating in acute retention prior to admission. His physical condition was very poor: there was a rigid chest with chronic emphysema and bronchitis for many years. There were great thirst, a dirty tongue, dull mentality and headache. He was put on permanent catheter drainage. By January 6, 1932, he had made a great improvement. The tongue was clean, the headache had disappeared, and there was a striking improvement in his general physical and mental deportment.

While we considered that his kidneys had now reached stability, we also knew that their reserve power must be very low, for he excreted no indigo-carmine in twenty minutes, and his urea concentration test results were never higher than 0·9%. Moreover, his chronic bronchitis was a dangerous factor. Many surgeons no doubt would consider it to be courting disaster to operate on such a patient. However, he came through the ordeal very well. Operation was performed on January 6, 1932, and at present he is up and about, with his wound practically healed.

CASE II.—W.H., aged seventy-four years, was admitted on September 5, 1931. He had a suprapubic puncture performed elsewhere for acute retention. He had all the signs of chronic urinary poisoning, and was much troubled with chronic arthritis. He was a "bad surgical risk". His urea concentration tests between September 7, 1931, and October 13, 1931, gave readings which never exceeded 1·9%. He excreted no indigo-carmine in thirty minutes. Again, this patient by any standard would be regarded as an extremely "poor risk". But he made great improvement in his general health on continuous catheter drainage, and by October 14, 1931, we considered that his kidneys had reached stability. The prostate was small and fibrous, and had to be removed with scissors and forceps. Throughout the operation his condition was excellent. He felt nothing, and slept the whole time. On return to bed he had a conversation and refused to believe that the operation was over. From then on his convalescence was uneventful.

CASE III.—M.M., aged seventy-five years, had a cerebral hæmorrhage sixteen years ago. He had had some paresis and motor aphasia ever since. There was a history of chronic urinary obstruction of about two years' duration. He was so feeble and so dull mentally that I was inclined to refuse operation at the outset. However, he was put on continuous catheter drainage, and at the end of six weeks had made such an improvement that I felt that operation was justifiable. He was operated on on December 4, 1931. At operation a hard fibrous prostate was enucleated with great difficulty; the patient felt no pain, and convalescence was uneventful. He was discharged on January 11, 1932. The striking thing about this patient was the extraordinary improvement in his general physical health and mentality as a result of prolonged catheter drainage.

I repeat again, one of the greatest advances ever made in the treatment of prostatic obstruction was the recognition that much of the patient's general ill-health is due to urinary poisoning from back pressure on the kidneys, and that by relief of this back pressure a great improvement in general health occurs. When stability is reached, by removing the prostate permanent relief is established.

I am sure that in local analgesia there is no shock and no added strain to any vital organs. In this way the great comfort of operative relief can be offered to these aged men with permanently impaired kidneys. To operate on men so far advanced under a general anæsthetic is courting disaster.

It would be tedious to give details of all our operation cases. We have had a sufficient variety to establish our faith in parasacral analgesia.

There was one difficult case of vesico-vaginal fistula in which operation was performed in perfect comfort and ease. Three weeks later the patient was given parasacral analgesia again to remove silkworm stitches high in the vault of the vagina. There were a number of patients on whom litholapaxy was performed and a number on whom fulguration was applied to papillomata of the bladder. For those extreme sensitive bladders affected with tuberculosis, cancer or interstitial cystitis, the anæsthesia was perfectly satisfactory.

Finally, we have observed in our prostatic operations that operative hæmorrhage is considerably less. Other observers have noticed this with sacral block anæsthesia. Lowsley and Rogers reported this finding as a result of 117 major urological operations performed under regional anæsthesia (*Journal of Urology*, November, 1924).

Conclusions.

We believe that of all forms of regional anæsthesia, parasacral is the best because the technique is simple, the percentage of failures few, and toxic and other reactions nil.

Operations of the lower urinary tract can be performed with no shock, little hæmorrhage and sufficient relaxation of muscles.

A combination of these advantages is to the betterment of our patients, no matter what the operation or however good a "surgical risk" they be. Such a combination is essential in operating on men with prostatic obstruction, and it allows us to offer permanent relief to aged and feeble men with severely damaged kidneys.

I am convinced that regional anæsthesia will allow us to offer operative relief to people who would be regarded as quite inoperable under general anæsthesia.

CONTINUOUS INTRAVENOUS THERAPY.

By H. M. Wilson and D. C. Moller,
Napier Hospital, New Zealand.

Continuous intravenous therapy has an accepted and definite value in the treatment of a variety of conditions associated with dehydration and toxæmia.

When fluid intake is greatly limited or impossible through the alimentary tract, continuous intravenous administration not infrequently provides a means of carrying the patient along until such time as sufficient intake by mouth is again possible. Its most brilliant achievement has perhaps been realized in abdominal surgical conditions, particularly in intestinal obstruction after late operations, and for the relief of serious post-operative sequelæ and peritonitis from any source.

The value of continuous intravenous therapy was amply demonstrated by Matas,[1], but, unfortunately, when he wrote the method of preparing the solutions and of keeping them at the correct temperature was somewhat cumbersome, and this rendered the use of this method in the average hospital a difficult matter.

Since then, Gallie has demonstrated that solutions may be given for long periods at room temperature; he has thus eliminated the necessity of cumbersome apparatus for maintaining a certain fixed temperature.

The great value of this therapeutic measure is emphasized by the ability to maintain a patient in good condition over a period often of one or more weeks, in spite of his inability to absorb fluid from the alimentary tract. Boothby, of the Mayo Clinic, has shown that one can fare very well and perform light work on an exclusive carbohydrate diet for sixty days at least; and it is not infrequent for a patient, as previously stated, to be kept alive for one or more weeks by the intravenous administration of a dextrose solution. After the technique of an administration had been rendered more practical by the work of Gallie, the only outstanding difficulty to be overcome was the tendency to reaction from the solutions used. In the past we have experienced many such reactions, some of which endangered the life of the patient; but since we have followed the technique outlined by Gallie and Harris,[2] we have had no reaction in the considerable number of patients treated in this way, although some hundreds of pints of fluid have been administered. The chief practical point in the technique advised was the discarding of the first portion of the distillate.

For many years, one of us was accustomed to use by subcutaneous route large quantities of fluid in the type of cases previously outlined, but the continuous intravenous method has solved the difficulties associated with: (i) sufficient quantity of fluid, (ii) the types of fluid necessary, (iii) the discomfort of the patient during the administration.

The following is an outline of the apparatus, technique and certain practical points in administration which we have found simple and serviceable.

The apparatus used is shown in the accompanying illustration. When a transfusion is given, use is made of the T-tube which is connected to a separate reservoir.

We have found it most convenient to use a vein in the leg, as this seems to cause the patient the least inconvenience, and permits the nursing of the patient to be unencumbered. In addition, we have followed Gallie's advice on the selection of as small a vein as possible. The one usually selected is that over

the internal malleolus. The cannula is inserted after exposure of a vein and tied into position. Sterilized gauze soked in 2% mercurochrome is packed into the wound surrounding the cannula, care being taken that the tissues are protected from pressure of the cannula by strips of gauze. The gauze is soaked daily with mercurochrome. The cannula and its attached tube are fixed to the leg by strapping. We have found that the insertion in the rubber system of a glass tube a few inches from the cannula has been of distinct value in enabling us to re-establish the flow after the giving of a blood transfusion. Not infrequently after the blood has been run in, it has been necessary to disconnect the tubing and reestablish the flow by means of an attached syringe before it is possible to reestablish the gravity flow of the saline solution and glucose.

In common with other observers, we have found that the cannula can be retained in the vein for three to five days, and that preceding the blockage in the veins the patient complains of local discomfort. It is our custom then either to make use of the vein in the other leg or to expose the same vein at a more proximal level.

We have found on some occasions that, although on incision of a vein blood flowed out, gravity flow would not permit the introduction of fluid through the cannula until many syringefuls of fluid had been introduced under pressure. In one case, this procedure had to be resorted to in place of the continuous gravity flow for some period of time, with a resultant phlebitis. As the quantity of fluid which has at times had to be introduced under pressure has been much greater than that which we would have considered necessary to dilate the collapsed veins, we have been inclined to regard the condition as a venous spasm.

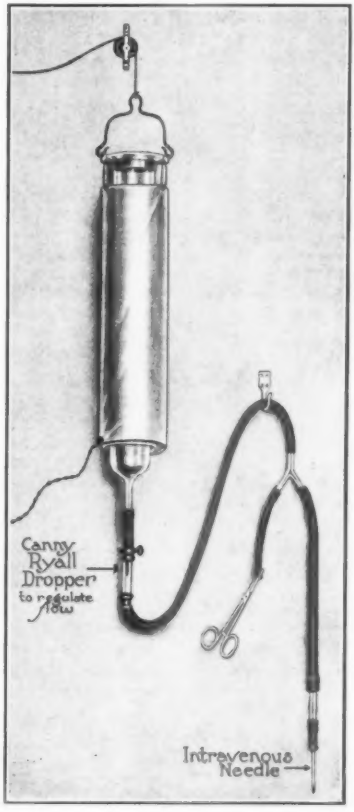


Figure showing apparatus used for continuous intravenous therapy.

Solutions.

The standard solutions we have used have been: (i) Normal saline solution, (ii) a 10% glucose solution, (iii) equal parts of normal saline and 10% glucose solution.

The glucose solution has usually been the isotonic 5% solution, except when a larger quantity of carbohydrate was needed; for example, in liver damage, when the 10% solution is used. The solution is poured into the reservoir at a temperature of 48·8° C. (120° F.), and the temperature maintained by means of an electric hot waterbag. The rate of flow is regulated by Canny Ryall's drip apparatus, as shown in the illustration, and at the commencement is usually two hundred to two hundred and forty cubic centimetres per hour; the rate is subsequently altered according to the patient's condition. At this rate, we have never seen the quantity of fluid produce symptoms or signs of vascular stress.

We consider it essential that a chart be kept of the total quantity of fluid administered in twenty-four hours, as well as of the patient's fluid loss by urine, vomited material and fluid motions. In addition, notice is taken of the state of the skin. By this means it is known whether sufficient fluid is being retained, and, when water excretion is limited, whether a reduction in the quantity of fluid given should be made. By these means such possible complications as œdema of tissues, water poisoning *et cetera*, can be guarded against to the maximum extent. When the dehydration of the patient has been sufficiently overcome, a reduction in the rate of flow can be made if a diminished urinary excretion suggests the possibility of a developing œdema. Our practice in regard to the use of insulin has varied. At first we were accustomed to administer one unit of insulin with every two grammes of carbohydrate. Recent work has shown that, in severe toxæmia, larger quantities of carbohydrate can be stored than under ordinary conditions. There is a transient rise of blood sugar when the intravenous flow is first commenced at the rate already outlined, but this usually returns to normal in about three hours. Recently, except in states of severe acidosis, we have not given insulin as a routine measure, unless examination of the urine at repeated intervals revealed glycosuria. Insulin is then added to the solution in the proportion mentioned. The continuous intravenous flow is maintained until a sufficient daily intake of fluid and carbohydrate is possible by the alimentary tract. The amount given intravenously is reduced approximately in inverse proportions to the increased quantity which the patient is able to take by mouth. The total time over which medication by this method is necessary naturally varies with the different types of conditions treated, and in one case the treatment was maintained for a period of ten days.

The following is an illustrating case.

Mrs. T., aged forty-six years, had cholecystectomy and drainage of the common duct performed for persistent jaundice. Five days after operation she commenced to bleed freely from the abdominal wound. Two transfusions were given on the two following days, and the wound was opened and packed. Incessant vomiting from gastric dilatation required continuous evacuation of stomach contents by the small nasal tube and the establishment of continuous intravenous therapy to combat the blood loss, dehydration and toxæmia. The following indicates the quantity of fluid administered daily.

First day: 5,760 cubic centimetres (192 fluid ounces) of equal parts of 10% glucose and saline solutions at the rate of 240 cubic centimetres per hour.

Second day: Same amount given.

Third day: 4,320 cubic centimetres (144 fluid ounces), 180 cubic centimetres per hour.

Fourth day: 2,100 cubic centimetres (70 fluid ounces), 150 cubic centimetres per hour *plus* 300 cubic centimetres (10 fluid ounces) of blood.

Fifth day: 3,420 cubic centimetres (114 fluid ounces), 140 cubic centimetres per hour *plus* 150 cubic centimetres (five fluid ounces) of blood.

Sixth day: 3,060 cubic centimetres (102 fluid ounces).

Seventh day: Intravenous therapy discontinued.

Tenth day: Intravenous therapy reestablished, 1,560 cubic centimetres (52 fluid ounces) in twelve hours.

Eleventh day: 3,840 cubic centimetres (128 fluid ounces).

Twelfth day: 3,720 cubic centimetres (124 fluid ounces).

Thirteenth day: Intravenous therapy discontinued permanently due to recovery of stomach function.

During the time over which this was carried on, four separate transfusions were given through the apparatus, in addition to three daily administrations of calcium chloride and two additions of sodium iodide.

We feel confident that this and other desperate cases associated with extensive loss of fluid by vomiting and marked cardiac asthenia, can be dealt with only

by a method which supplies fluid and nutriment intravenously, slowly and continuously.

In these desperate cases we regard it as a life-saving measure. We put forward this article with a knowledge that it contains nothing original, but with a plea for the more frequent use of the method in these cases of abdominal disorder, dehydration and toxæmia.

References.

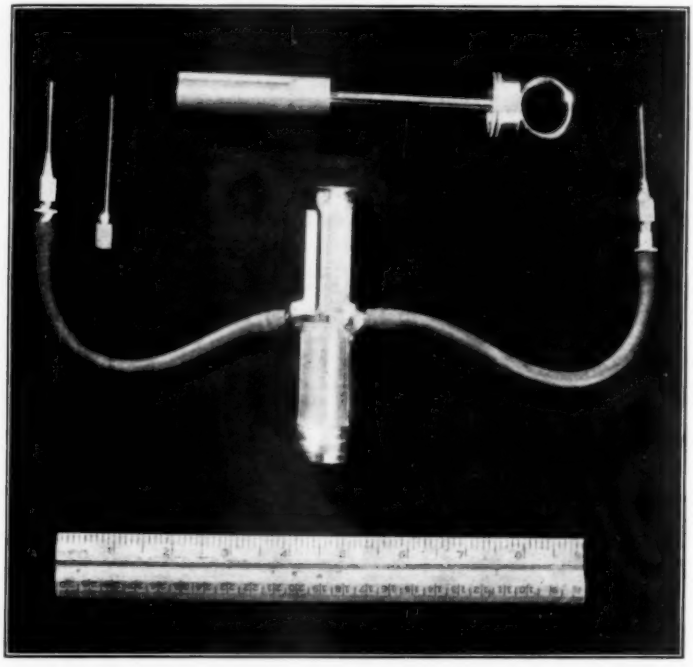
(1) R. Matas: "The Continued Intravenous 'Drip', with Remarks on the Value of Continued Gastric Drainage and Irrigation by Nasal Intubation with a Gastro-duodenal Tube (Jutte) in Surgical Practice", *Annals of Surgery*, Volume LXXIX, 1924, page 643.

(2) W. E. Gallie and R. I. Harris: "Continuous Intravenous Administration of Physiological Salt Solution", *Annals of Surgery*, Volume XCI, March, 1930, page 422.

TRANSFUSION OF WHOLE BLOOD BY THE JUBÉ SYRINGE.

By Douglas Robb,
Auckland.

The Jubé apparatus consists, as shown in the photograph, of a glass syringe barrel with two lateral tubes. To each of these is attached an 18 centimetre rubber tube. One rubber tube fits on to a wide bore needle for the donor's vein.



Blood transfusion syringe by Louis Jubé. Trocar and cannula on left for recipient; large needle on right for donor.

The other fits on to a cannula of one millimetre internal diameter for the recipient's vein. The piston is half as long as the barrel, and has a groove on one side which reaches just above the level of the lateral tubes when the piston is fully inserted. With the groove opposite the donor's lateral tube, the piston is

withdrawn and the syringe is filled with blood. The piston is then rotated through 180° until the groove is opposite the recipient's lateral tube. On pushing down the piston the contents of the syringe are now discharged into the recipient's tube. Each cycle of withdrawal-rotation-insertion discharges 10 cubic centimetres (or five cubic centimetres in the smaller model), and in a few minutes any quantity of blood up to 500 cubic centimetres or more can be transfused. The instrument is accurately made, and the alterations in calibre between needle, connexions, rubber tubes and cannula are made gradually.

In practice the syringe and fittings are sterilized with methylated spirits. The parts are connected up, and spirit and afterwards sterile liquid paraffin are syringed through the whole system. The piston is then removed and separately dipped in liquid paraffin. This is an important detail, as it diminishes the chance of blood sticking between piston and barrel and thus interfering with the transfusion. Suitable veins in donor and recipient are displayed by tourniquets, and the parts approximated. The recipient's vein is approached first, as it is usually the more difficult to puncture. In a favourable case it can be punctured direct with trocar and cannula. Sometimes it is necessary to cut down on the vein under local anæsthesia, and insert the cannula after nicking the wall of the vein with scissors. When the cannula is correctly placed the trocar is partially reinserted, and attention is given to the donor. Usually the needle, attached by tube to the syringe, is inserted without difficulty, preferably pointed distally in the median basilic. Blood flows freely and the syringe is filled. By holding the syringe so that the recipient's side of the tube, with rubber and metal connexion fitted, is uppermost, any air and residual paraffin are expelled first on pushing the piston home. When pure blood flows, the recipient's trocar is removed, the tube attached to the base of the cannula and the transfusion proceeded with.

With practice these manœuvres can be quickly and surely carried out, and up to 500 cubic centimetres of warm, pure, unaltered blood can be transfused in a few minutes. The method is particularly suited to medical cases where repeated small transfusions are called for, but is very generally useful in ordinary surgical work.

I have found one mechanical defect in the syringes used in Auckland. The glass barrel is easily cracked at the upper end when inserting the heavy metal piston. This does not occur if the instrument is handled carefully, but if the defect were remedied by the fitting of heavier glass barrels, the apparatus would be not far short of perfect for its purpose.

Case Reports.

EVENTRATION OF THE DIAPHRAGM.

By P. Stanley Foster,
Christchurch.

Miss B., a schoolgirl, aged fourteen years, had previously been healthy and well, taking a normal part in school life and games. She was seen in consultation after two attacks of lower abdominal pain, and, on examination, an easily palpable tumour was discovered above the pubes. Operation was performed and the tumour

Figure I.

proved to be a large ovarian cyst, which was duly removed. In searching for the appendix in the routine examination of the abdomen, it was then found that the liver was lying lower than usual and below it no small intestine could be seen. The stomach appeared normal, but the duodenum ran sharply up under the liver and disappeared. Close to it appeared the transverse colon (also from under the liver), which ran to the left and continued normally. On attempting to pass the hand up over the anterior surface of the liver, a peritoneal attachment was encountered, barring the way. Posteriorly no pouch could be found. The gall bladder was not seen. During convalescence, a complete radiographic examination was made with barium meal and enema.

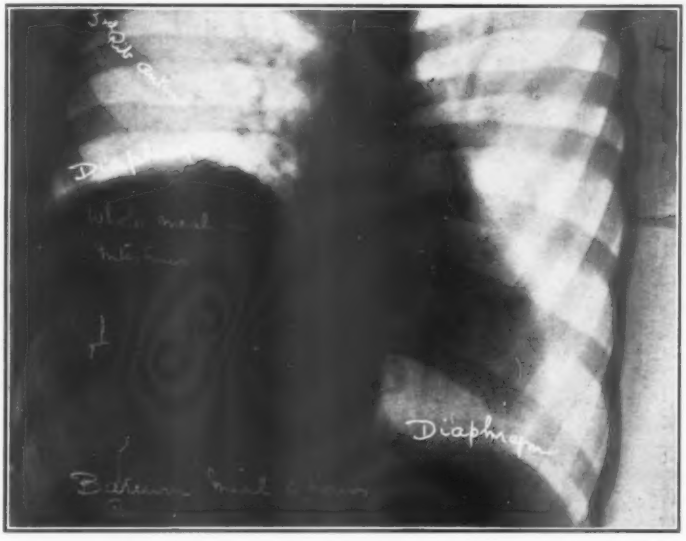


FIGURE II.

Figure I shows the meal in the stomach (after two hours) and in the small intestine. The upper level of liver is marked with lead. The duodenum is well shown. Also a portion of small intestine can be seen in lower part of the right side of the chest.

In Figure II the whole meal is well shown at six hours in the right side of the chest. The diaphragm is seen between the third and fourth ribs anteriorly.

In Figure III a barium enema shows the caecum above the liver and also the disposition of the remainder of the colon.

Figure IV is a drawing of the condition.

The case is, I think, one of eventration of the diaphragm, a condition of elevation of one side of the diaphragm described by Petit in 1774. Cruveilbier used the word "eventration" in 1829. Radiology has disclosed cases in increasing numbers, though Jenkinson, in the *American Journal of Roentgenology*, states that only three cases are described on the right side and that only one was diagnosed before death. A. J. Walton, in the same journal, in 1924, states that six cases of right-sided eventration have been reported, but adds that the liver always fills up the projection.

FIGURE III.

This case seems to be one of an undoubted right-sided eventration *plus* an unusual congenital abnormality. It is interesting to speculate, first, upon what will happen if the patient develops appendicitis, and, secondly, upon the state of the biliary passages, as I did not localize her gall bladder or pancreas.

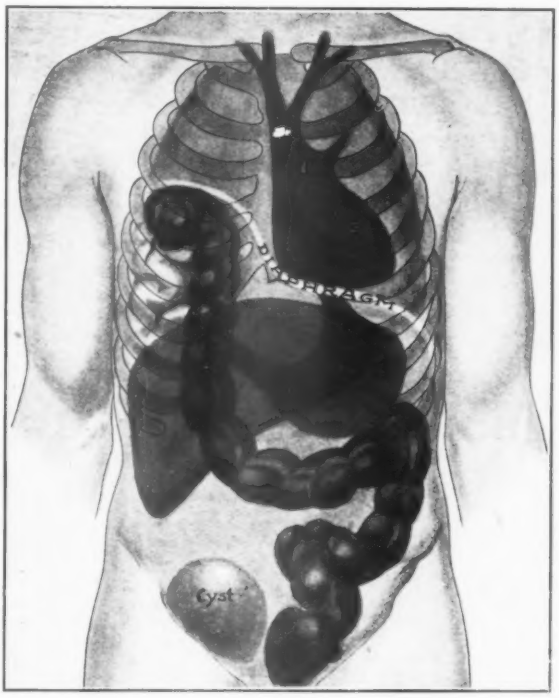


FIGURE IV.

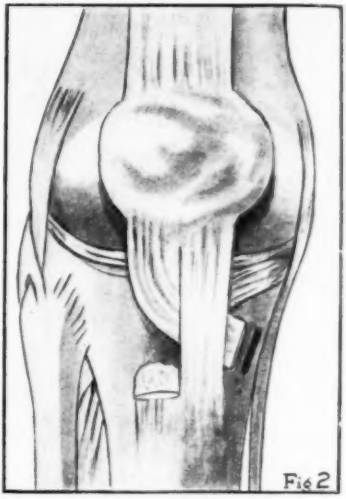
Acknowledgement.

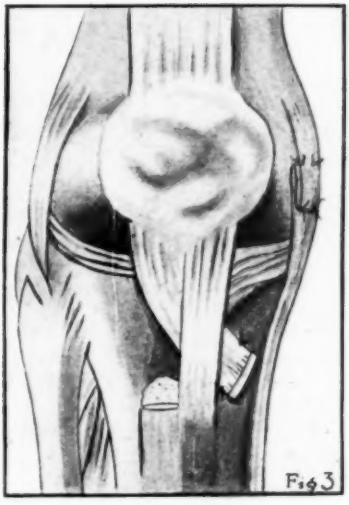
I am greatly indebted to Dr. Neil Guthrie and his X Ray Department at the Christchurch Hospital for the excellent radiograms and for help afforded from his radiological journals.

RECURRENT DISLOCATION OF THE RIGHT PATELLA.

By K. S. MACKY,
Auckland.

A GIRL, aged fourteen, was admitted to hospital under my care on July 10, 1930, with the history that for some six years the right knee cap had been slipping on to





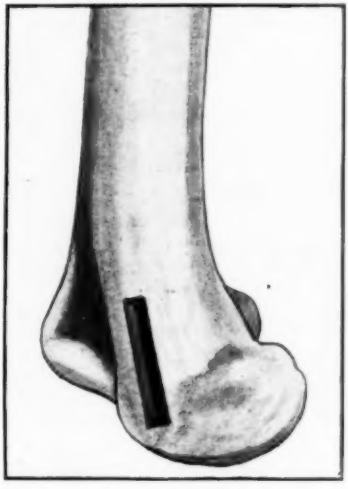


FIGURE IV.

the outer side of the knee whenever she fully extended the leg. The left knee cap used to do likewise, but had been stable for some years. There was no history of injury. On examination, the patella was abnormally mobile and placed high on the condyles, the *ligamentum patellæ* being elongated. The anterior surface of the condyle did not appear to be lower than normal. No valgoid deformity of the knee was present.

On July 22, 1930, I performed the Goldthwaite operation, dividing the *ligamentum patellæ* longitudinally and separating the lateral half off with a piece of bone at its attachment. This was then passed beneath the medial portion, pulled taut, and sutured to a slit in the sartorius expansion, the underlying bone being freshened to receive the piece of bone attached to the ligament. The after-treatment consisted of keeping the knee for six weeks in extension with, after two weeks, gentle surging faradism to the *vastus internus*, and gripping exercises at the end of a month. No flexion was allowed until the end of the six weeks.

About the end of August the girl returned and reported that the knee cap had slipped out again. On September 30, 1930, further reconstructive work was carried out, and included osteotomy of the lateral condyle, with elevation of the anterior fragment and the insertion of a wedge of bone removed from the tibia into the gap. Even this did not seem sufficient, and it was not until the capsule, on the inner side, was plicated that stability was obtained. The operation is depicted in the accompanying illustrations.

It is now over a year since the last operation, and the girl has full movements of the joint and is playing games.

In this case, I think the cause of the disability was the elongated *ligamentum patellæ*.

CARBUNCLE OF THE KIDNEY.

By HELEN MAYO AND G. H. BURNELL,
Adelaide.

ALTHOUGH the term "carbuncle of the kidney" has been in use for many years, the recognition of this condition as a clear-cut clinical entity does not appear to have been general. The name is applied to an acute or subacute infection of the renal parenchyma by the *Staphylococcus aureus*, which leads to the development of one or more necrotic areas in the renal tissues similar in appearance to that of a carbuncle in the skin. The necrotic area has a great tendency to perforate the renal cortex, leading to the development of a perirenal abscess, but it usually does not invade the renal pelvis until very late in the disease, so that the urinary findings, for a considerable time after the onset of the disease, are normal.

The organism is derived from some staphylococcal infection elsewhere in the body, for example, a carbuncle of the skin, a boil, or a whitlow, but may also arise from osteomyelitis, a dental abscess or similar septic focus.

The pathological picture presented by the kidney is different from that of pyæmic foci, which are usually numerous and scattered throughout the renal tissue, whereas in carbuncle the lesion is usually single, or at most consists of two or three separate foci. Dick[1] states that only twenty-seven cases of this disease are reported in the literature, and, if this be so, one can conclude that the true nature of such cases has been overlooked. Dick also states that the earliest age of onset is ten years, so that the present patient is the youngest one yet recorded.

S.C., a girl, aged nine years, was admitted to the Adelaide Children's Hospital on May 21, 1931. Her history showed that an attack resembling influenza had begun four days before, for which she was confined to bed. The day after onset her nose had bled. On the day prior to admission her ankles swelled and became

painful, so that she was unable to move her feet. There was no shivering attack. She had vomited several times during the first three days. There had been slight frequency of urination, but no dysuria. She had had an attack of measles six months before admission to hospital. When she was admitted, her temperature was 38·7° C. (102·8° F.), the pulse rate was 104, and respirations numbered 24. A general examination disclosed no abnormality except the swollen and painful ankle joints. The apex beat of the heart was just outside the mid-clavicular line, and there was no bruit. The urine was normal.

Salicylates were administered, and on the seventh day of illness her temperature was normal, and she appeared quite well. This improvement continued until June 2, 1931, when her temperature rose to 37·3° C. (99·2° F.) and a slight impurity of her first cardiac sound was heard. Her temperature continued to show some slight irregularity with a maximum of 37·6° C. (99·8° F.) until June 19, 1931, and there was a definite systolic murmur. So far her condition presented no unusual features. On June 19, 1931, some pus was detected in the urine, which on culture gave a pure growth of *Staphylococcus aureus*. The next day her temperature rose to 39·5° C. (103·2° F.). Hexamine was administered, and improvement followed for some six days. On June 27 the temperature again rose, and on June 30 reached 40° C. (104° F.). At this time the condition of the child showed a profound change for the worse. She appeared extremely ill, the sunken eyes, pinched face, dry skin and grey colour indicating clearly her dehydrated and toxic condition. An erythematous rash in the left axilla and on the left thigh made its appearance.

Examination of the urine showed pus and a coliform bacillus on culture. Some fullness in the right loin was noted. Attempts at blood culture on July 3, 1931, and July 16, 1931, were without result. Administration of alkalis and other medical measures were of no benefit, and on July 17, 1931, she was seen by one of us, who made the following observations:

The patient was very ill and wasted, the tongue dry and furred, the temperature of the hectic type, swinging widely each day. There was marked tenderness and rigidity, almost board-like, of the muscles both front and back on attempting to palpate the right kidney. It was thought that a swelling could be felt in this situation. There was little complaint of pain, although the tenderness was so marked.

Cystoscopy and bilateral pyelography were done on the same day. The bladder and ureteric orifices were normal. Specimens of urine were collected from both kidneys. That from the right side gave a culture of coliform organisms, while that from the left was clear. The pyelogram showed a normal left renal pelvis and calyces, but on the right side the opaque medium had assumed an indefinite shape to the outer side of one solitary calyx, which had been filled. The radiologist reported a perirenal abscess present (see Figure I). On July 20, 1931, a definite swelling was visible in the right loin, and there was now considerable pain in this region.

The swelling was incised, and the perinephric abscess evacuated. Owing to the bad general condition of the patient, nothing further was done at this time. A culture of the pus resulted in a pure growth of *Staphylococcus aureus*. After the opening of the abscess, the child's general condition improved, but the temperature continued to swing widely, and there was very little discharge from the wound. On July 30, 1931, under nitrous oxide and oxygen anæsthesia, the original incision was enlarged, and the kidney exposed by the muscle-splitting method. Stripping of the organ from its bed was very difficult, owing to the thick-walled abscess cavity present, and while it was being done, the pleural cavity was opened with the finger. This opening was immediately closed, and the kidney delivered. Near its upper pole the surface of the organ was irregularly nodular, and there were five or six small openings from within on to the surface, through which openings pus was oozing freely. At the lower pole were similar nodules, but these had not perforated. The kidney was removed, and the wound closed, a rubber tissue drain being inserted.

Her convalescence was rather stormy, and the temperature remained high for seven days after operation. It was thought that an empyema was developing, and exploration of the right pleural cavity with a needle yielded purulent fluid, but a day or two later there was a profuse discharge from the operation wound, and the temperature subsided; apparently the pleural collection had discharged itself in this direction. From then on her recovery was uneventful, and she was out of bed by September 1, 1931. No cardiac bruit could be detected at this time.

On sectioning the kidney after removal, it was seen that there were two necrotic areas in the parenchyma, one situated at the junction of the middle and

FIGURE I.

upper thirds, and impinging on the upper part of the pelvis, and another at the lower pole just beneath the cortex (see Figure II). The upper lesion, as before stated, had ruptured through the capsule of the kidney at several points, giving it a moth-eaten or pitted appearance; no point of communication with the renal pelvis could be seen. The lesions macroscopically bore a certain resemblance to tuberculous disease, consisting of ragged collections of pus and necrotic tissue, but the margins of the lesions were rather more sharply defined than is usually the case in tuberculosis.

Sections of the kidney by Dr. F. R. Hone revealed two chronic abscesses of the kidney, and staphylococci were demonstrated in the sections at the edges of the lesions. That the process was of fairly long duration was evident from the fact that there had been formed a good deal of young fibrous tissue about the edges. Sections taken at some distance from the lesions showed no abnormality except cloudy swelling.

Comment.

It is difficult to see how the original diagnosis of subacute rheumatism can be set aside, though in the absence of specific tests for rheumatism, it may not be possible to prove its presence. Since the time of Bouillaud the coincidence of painful swollen joints, raised temperature and cardiac involvement has been associated with subacute rheumatism, and as far as can be ascertained, they have not been present in other cases of carbuncle of the kidney. When, however, one considers the case in the light of subsequent events, one is forced to the conclusion that the suppurative process in the kidney probably began about the time of the onset of the illness.

The absence of local symptoms in the early stages is unusual, as one of the first complaints, in reported cases, is of pain in the side. The age of the child and her confinement to bed in a hospital ward may account for this, as the absence of active movement may have minimized the pain, and the absence of a confidant may have prevented its disclosure.

No causal focus was found, but the presence of a staphylococcal urinary infection, with slight pyuria, combined with such severe toxic symptoms, should have led us to consider the diagnosis of carbuncle of the kidney at an earlier stage of the illness.

Acknowledgement.

Our thanks are due to Miss L. Hannaford for the coloured plate.

Reference.

(1) B. M. Dick: "Staphylococcal Suppurative Nephritis (Carbuncle of the Kidney)", *The British Journal of Surgery*, Volume XVI, July, 1928, page 106.

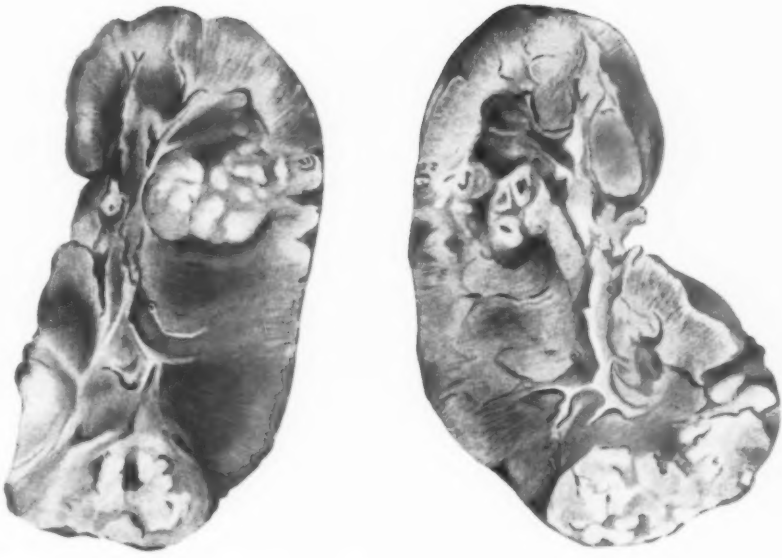


FIGURE II.

Surgery in Other Countries.

[In this column will be published short résumés of articles likely to be of practical value from journals published in other countries and not readily accessible to surgeons in Australia and New Zealand.]

Phalangization of the First Metacarpal Bone.

The technique, indications and results of the phalangization of the first metacarpal bone are discussed by P. Bonnet and F. Carcassonne in *Revue de Chirurgie*, May 5, 1931.

This desirable result is not easy to attain. Since the thumb is the principal member of the hand, in virtue of its movements of opposition, any operation which will restore this valuable digit or a modification of it, is a welcome addition to surgery.

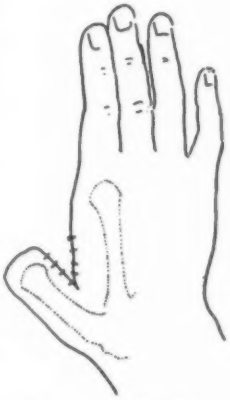


Figure I. Showing scheme of completed operation.

Bonnet and Carcassonne recommend the incision first described by Huguier. This incision runs parallel with the first metacarpal bone and divides the web extending on the ventral and dorsal aspects to within 1·25 centimetres (half an inch) of the base of the bone. Instead of removing the dorsal interosseous and the transverse head of the *adductor pollicis* muscles, they prefer to detach the adductor with the sesamoid bone from its insertion and to implant it as low as possible on the postero-medial aspect of the base of the first metacarpal.

The method of fixation is by means of a loop of catgut. It is not necessary to perforate the metacarpal bone to insure the proper fixation of the sesamoid to it.

The closure of the wound is not easy. In many cases there are scars on the hand binding down the skin, and even with good flaps it is essential to have no tension; thus skin grafting of the raw area remaining after section of the web may be required.

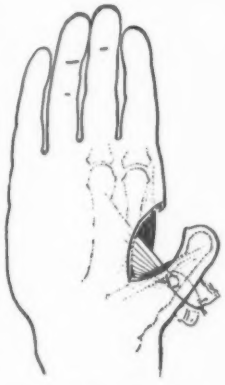


Figure II. Showing detachment of adductor muscle and its implantation into the first metacarpal bone.

The pre-operative preparation of the hand for plastic work of this kind is most important. It is not essential to have the wounds of the hand, due to trauma maybe, completely healed, but the hand must at least be disinfected for some days. It may be bathed in Dakin's solution or in a solution of potassium permanganate for several days before operation. The authors prefer an iodized alcohol dressing from the elbow to the finger tips as a preparation for several

days; also an injection of antitetanic serum is given before the operation is commenced. A tourniquet is not used, but fine catgut ligatures are applied where required. After completion of the operation and the application of dressings, the hand is put up in plaster with the reconstructed thumb in a position of abduction and slight opposition. Active and passive movements are commenced on the eighth day, and on the fifteenth day warm baths are employed to soften the scar. From then on the patient freely uses the new digit.

Modifications of this technique may be adopted where indicated. If, for example, the index finger has been amputated as well as the thumb, the second metacarpal bone may be removed, a wider space thus being permitted between the first metacarpal (now mobilized) and the remainder of the hand. There are objections to this procedure, however, and it should not be adopted unless specially indicated by reason of the patient's work *et cetera*.

FIGURE III. Showing condition of hand after operation.

Contraindications to the operation are the absence of a complete first metacarpal bone, severe damage to the adductor muscle, paralysis of that muscle, arthritis or ankylosis of the carpo-metacarpal joint of the thumb. A further consideration is the psychological attitude of the patient. If she or he insists on an æsthetic result, the operation is best not attempted, since the result will be a functional one only. The mutilation will persist. The digit which is liberated is short, and many surgeons consider this a serious objection to the operation, but a prosthesis may be fitted to enable the patient to obtain a better grasp of small objects.

Bonnet and Carcassonne are enthusiastic in recommending this operation. They have performed it on two occasions with good results. Twenty-seven cases are collected from the literature with three excellent results, twenty-two good results, and two moderately good.

For full operative details, this article should be read in conjunction with an excellent account of an operation for the reconstitution of the thumb by J. Leonard Joyce, *The British Journal of Surgery*, January, 1929.

The reviewer has employed the Huguier method in one case, that of a female who lost all her digits in a sausage machine. The first metacarpal was mobilized and a useful hand was obtained, but, being a woman, the patient was more concerned with the appearance than with its functional value. She is able to write and do all kinds or work requiring simple grasping of small objects, but she still furtively conceals the injured member whenever possible. However, there is no doubt that any kind of prehensile digit is preferable to a thumbless hand.

A. E. COATES.

EXPOSURE OF THE TEMPORO-MAXILLARY JOINT.

IN *Der Chirurg*, August 15, 1931, Professor Axhausen, Director of the Jaw Clinic in The Charité, Berlin, advocates strongly a retro-auricular approach of the temporo-maxillary joint, condyle and neck of the inferior maxilla. Axhausen emphasizes the difficulties and disadvantages of the anterior or pre-auricular approach. These are the result of the limited space, the risk of damage to the facial nerve, the depth of the wound, and the difficulty of hæmostasis.

Axhausen favours a modification of the retro-auricular approach first described by Bockenheimer. Bockenheimer's method, though free from the disadvantages

and difficulties ascribed to the pre-auricular approach, and though it gives an excellent exposure of the joint, condyle and neck of the maxilla down to the region of the coronoid process, has been the subject of adverse criticism. It has been contended that it is difficult to secure efficient sterilization of the field for

FIGURE I.

FIGURE II.

operation, and that it is difficult to secure healing without a narrowing of the auditory meatus, and consequent interference with hearing.

Axhausen, as a result of experience in his clinic (where surgical exposure of the temporo-maxillary joint is a frequent necessity), finds that sterilization of the external auditory meatus can be carried out effectively, provided the ear and external auditory meatus are healthy. He has no doubt, however, that narrowing

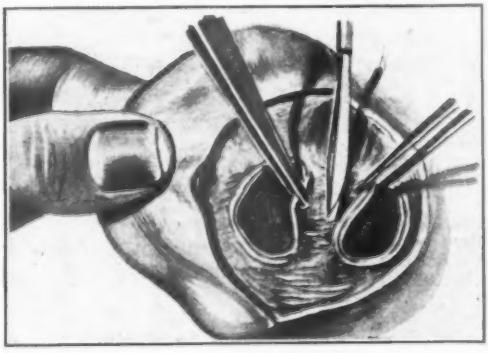


FIGURE III.

of the auditory meatus and auditory troubles may follow as a result of delayed and inexact union of the cartilaginous layer of the meatus and the accompanying scar tissue formation. He has shown that this occurs notwithstanding the use of various mechanical aids.

Axhausen's modification of Bockenheimer's method eliminates the risk of any narrowing of the auditory meatus by an incision which divides the cartilage in the *cavum conchæ* (see Figure I) only, the cartilage of the tragus and anterior wall being retracted. Preparatory treatment should be preceded by a thorough aural examination carried out by an aural specialist, in order to exclude any infective *otitis media*.

Sterilization is effected by instillation of oil, syringeing, cleansing with alcohol, and the application of iodine. At the end of the process of disinfection, the meatus is closed by means of a sterile tampon which is retained until the end of the operation.

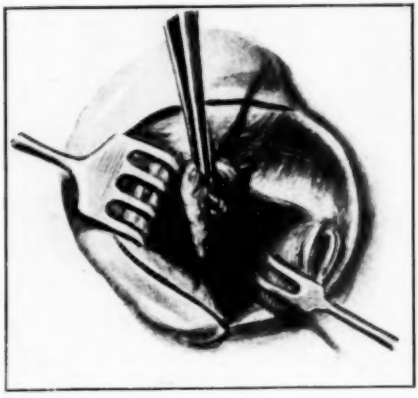


FIGURE IV.

The steps of the operation are most readily followed by a reference to the accompanying illustrations, which have been taken from Axhausen's papers. Figure I shows the incision around the opening of the external meatus; posteriorly the incision extends into the *cavum conchæ*. Figure II shows the second step. The pinna is pulled forward and an incision is made from its upper to its lower border. The cartilage is divided through its mid-portion, and thus exposure of the anterior incision is obtained. Division of the cartilage around the meatus is completed above and below. Figure III shows the isolation of the cartilaginous structures of the meatus above, anteriorly and below. Posteriorly their attachments to surrounding tissues are not interfered with. Figure IV shows the separation of the soft parts by means of sharp and blunt dissection, and their retraction forward; a fair-sized vessel (see Figure V) generally requires ligating. The soft parts include a thin layer of the parotid. Occasionally a thin layer of the parotid spreads back around the posterior border of the maxilla, and, if this is the case, it may be divided without likelihood of the occurrence of any troublesome hæmorrhage. Figure V shows the exposure of the region of the temporo-maxillary joint.

If desirable, the neck and upper portion of the ramus can be readily exposed by a continuation of the incision along the posterior border of the condyle and ramus, and the blunt dissection of the soft parts forwards.

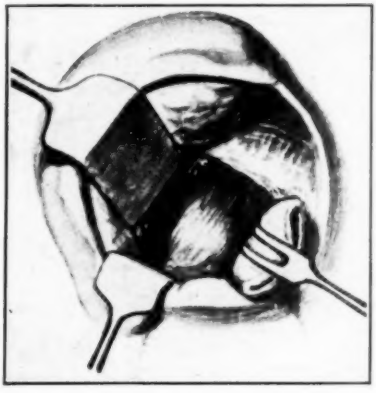


FIGURE V.

At the end of operative interference with the joint, the ear is turned back. It readily falls into its proper position, and only sutures passing through skin and subcutaneous tissues are necessary to insure alignment.

Axhausen, at the time of writing, had employed this method in sixteen cases. All wounds healed *per primam*. Contraction of the auditory meatus is not

possible, and the resulting scar was invisible. Axhausen points out that the operation is followed by temporary loss of cutaneous sensation over the pinna; recovery of this sensation is generally established in from three to four months.

B. T. ZWAR.

THE SURGICAL TREATMENT OF ANGINA PECTORIS.

THE fact that of the 2,943 medical men in the United States who died in 1930, 1,059 succumbed to cardiac affections, and of these 109 perished in the agonies of anginal attacks, is sufficient reason for summarizing the results of surgical interference in *angina pectoris*. Records of 100 patients operated on prior to 1925 have been analysed by R. Leriche and R. Fontaine in *Journal de Chirurgie*, December, 1931, and a further 78 cases since that date have been collected by them.

The series of 1925 showed 30·8% of good results for over four months, while the recent 78 cases yield 33·3% of good results for over one year. The authors state definitely that the surgical treatment of angina is *un fait accompli*. In some cases the attacks are completely arrested, in others minor crises persist, but the severe attacks are absent. The best results are obtained in patients with a minimum of organic lesions. The immediate mortality in the 1925 series was 13·8%, and in the recent 78 cases 14·2%. After discussing the physiological basis of the operations as worked out by François Franck, the authors conclude that the removal of the stellate ganglion is the correct operation.

The operations of Jonnesco and Danielopolu are critically examined and the difficulties and dangers of such procedures are emphasized. The objections of these two writers to removal of the stellate ganglion are regarded as having no experimental foundation. Their bad results are stated by Leriche and Fontaine to have been due to faulty technique and rough handling of delicate structures. An analysis of the fatal cases reported by other writers is given.

The operation of resection of the stellate ganglion is performed in the neck. The resection of ribs is not necessary as when the sympathetic is approached from the back. Minor operations on the cervical sympathetic, such as removal of the superior cervical ganglion, which do not produce the desired effect, should not be done, since it is more difficult to obtain relief later by stellate ganglion removal. Such operations may produce reflex disturbances of the cardio-accelerator mechanism without relieving the anginal pain, and later operations on the stellate ganglion do not appear so successful.

Leriche does not accept Langley's view of the sympathetic nervous system, but adopts the ideas of François Franck in regarding the sympathetic ganglia, especially the stellate, as reflex centres. He looks upon angina as a sympathetic reflex, the impulse arising in the cardio-aortic area and relaying in the stellate ganglion *vid* the coronary vasomotor fibres, but diffusing also to the medullary centres by way of the *rami communicantes*. On this theory it appears desirable to suppress the reflex centres, and this is done by removing the stellate ganglion. Leriche states candidly that he does not know exactly what Danielopolu actually does in his operation (most English readers will agree with this), and he concludes from his experimental and clinical work that the simple removal of one or both stellate ganglia is the best operation. The left ganglion should be first removed after the feasibility of the operation has been tested by preliminary injection of "Novocain" into the ganglion region during a crisis. Should the attacks not be relieved by the unilateral operation, the right ganglion is then removed some weeks or months later.

The following are the reports of cases collected to date. The results of the bilateral operation in six cases are as follows: Two patients were cured for over one year, three were cured for less than one year, in one a failure occurred. The good results were thus 83%.

For the unilateral operation in 18 cases the following results are given: Three patients were cured for over one year, six were cured for less than one year. There were five failures. In the remaining cases post-operative fatalities occurred.

Death in the fatal cases is probably due to irritation of the stellate ganglion during operation. Autopsy revealed two cases of coronary thrombosis, two of pulmonary œdema. The results after removal of the superior cervical ganglion on one or both sides are also given, together with the figures obtained by Danielopolu in forty-five cases.

The mass of statistics, however, does not give much information of value since the type of patient operated on, the severity of the disease and the late results are not mentioned.

Leriche and Fontaine's results have been obtained by operating on selected patients with the most careful technique. Their operation has been performed in many cases in which the extra-stellate operations failed. Though the number of cases is small, the results seem to be encouraging. Leriche maintains that relief is more definite after stellate ganglion removal than after any other operation on the sympathetic nervous system. Time alone will show what may be expected of surgery in these distressing cases. Further communications from the Strasbourg Clinic will be welcomed.

A. E. COATES.

EXTRASPHINCTERIC RECTAL FISTULA.

J. PETERMAN, of the Surgical Department of St. Hedwig's Hospital, Berlin, writing in *Der Chirurg*, Volume III, page 552, describes a new method of dealing with extrasphincteric rectal fistulæ. He acknowledges the previous work of Kleinschmidt and Moszkowicz.

After stressing the ineffectiveness of non-operative measures, he lays down the first requirement of safe operation as: "No damage to the sphincters." He does not agree that a single division of the sphincters is more than relatively safe, as regards future incontinence, and stresses the danger, in respect to incontinence, of hurried or forceful dilatation. He insists on slow dilatation, preferably under local anæsthesia. Damage to the *levator ani*, necessary in cases in which the fistula penetrates the pelvic diaphragm, does not produce incontinence.

He emphasizes the ease of treatment in intrasphincteric fistulæ, contrasted with the difficulty of the extrasphincteric type, and in regard to the latter stresses the fact that the success or failure of the operation hinges on the successful localization and closure of the bowel communication, all other contributing factors being of comparatively negligible importance. He achieves the localization by means of the injection of glycerin solutions of methylene blue, these being less diffusible than watery solutions. The principle of his operation is the conversion of the extrasphincteric into an intrasphincteric fistula. The steps of it are: (i) A bow-shaped modification of Sudek's "V" incision. (ii) A careful dissection out and removal of the whole fistula tract and of its ramifications, without injury to the sphincters. (iii) Mobilization of the rectal wall round the opening, with the careful removal of callous or granulation tissue in this neighbourhood. Where possible, the rectal wall is drawn down and fixed so that the old fistula opening lies one to two centimetres lower than it did before. (iv) The formation of a canal between the sphincter and the anal lining up to the old fistula mouth by means of an incision in the posterior muco-cutaneous junction, and by careful boring upwards with the forceps; and the laying of a tube the thickness of a finger in the canal so formed. (v) Closure of the wound outside the sphincter, completely by suture if possible, special care being given to the tissue in proximity to the bowel wall, and no dead spaces being left. If it cannot be closed thus, the inlaying of a fat graft after Payr's method, or the covering of the skin defect by pedicled skin grafts according to Moszkowicz. (vi) Three to four weeks later the tube is removed and the track is guttered as in an intrasphincteric fistula. Peterman cites five years' experience of the method; healing always occurred and full continence resulted.

A. E. BROWN.

Reviews.

PRE-OPERATIVE AND POST-OPERATIVE TREATMENT.

"Principles of Preoperative and Postoperative Treatment", by Reginald Alex. Cutting, is the second of a series of surgical monographs published by Paul B. Hoeber.[1]

Dr. Rudolph Matas in a very interesting foreword states the keynote of the work in the following apt way: "The surgeon whose interest in his patient starts with the knife and ends with the safety pin" has no place in the hierarchy of surgery.

Increased biochemical knowledge and other aids such as the electrocardiograph and better understanding of the underlying pathology of many lesions have all imposed new duties and new responsibilities on the operating surgeon. The old jibe about "a laboratory diagnosis" has lost a lot of its sting, for it is now realized that special investigations often save a life to the patient and add to the reputation of the surgeon.

Dr. Cutting's book will fill a definite void in medical literature, as he has selected from the medical literature of the last fifteen years, which might be called the era of biochemistry in surgery, all that relates to the better pre-operative preparation and the post-operative care of the patient, and has given it to the profession in one well printed, carefully written and easily digested volume.

"Blood Transfusion", "Water Balance and Dehydration", "Disturbances of Acid-base Equilibrium: Acidosis and Alkalosis", "Abdominal Distention and Gas Pains", "Dilatation of the Stomach", "Urinary Disturbances", "Postoperative Pulmonary Complications" and "Thrombophlebitis", are all treated in separate chapters and in each chapter the author presents a very satisfactory *résumé* of present day surgical opinion.

At the end of each chapter is appended an extensive list of references for the gratification of the appetite which the author's views create.

The last section of the book, dealing with the especial care of patients suffering from gastric, prostatic, thyreoid, cranial and intestinal diseases, is by no means the least interesting and instructive. Every hospital and medical school library should contain a copy of this most instructive work.

THYREOID DISEASES.

Cecil A. Joll in a readable volume of moderate size presents his views on thyreoid diseases based on an extensive experience gained at the Royal Free Hospital, London, and in his private practice.[2] As the author states in his preface, no British work attempting a general survey of thyreoid diseases has been published for over thirty years so that there is need for such a volume as the one under review.

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[1] "Hoeber's Surgical Monographs: Principles of Preoperative and Postoperative Treatment", by R. A. Cutting, M.D., C.M., M.A., Ph.D., with foreword by Rudolph Matas; 1932. New York: Paul B. Hoeber. Imperial 8vo., pp. 832, with 76 illustrations. Price: $10.00 net.

[2] "Diseases of the Thyroid Gland, with Special Reference to Thyrotoxicosis", by Cecil A. Joll, M.S., B.Sc., F.R.C.S.; 1932. London: William Heinemann (Medical Books) Limited. Crown 4to., pp. 700, with illustrations. Price: 63s. net.

In the first chapter the author deals with the structure, development of the thyreoid gland, its relation, and its blood supply. The second chapter is devoted to thyreoid physiology. Mr. Joll then presents his classification of thyreoid diseases and proceeds to discuss each group in turn.

In his discussion of the development of the organ he agrees with those who affirm that it arises from a mid-ventral pharyngeal diverticulum, and that the fourth and fifth bronchial arches add nothing to the adult thyreoid in man. In passing he fully discusses the embryological explanation of the various features of thyreoglossal cysts, following closely the well known views of Hamilton Bailey; and as regards their removal he favours the methods of Sistrunk.

One of the most interesting chapters is that devoted to the consideration of the ætiology and geographical distribution of simple goitre.

The various types of dysfunction of the gland and their appropriate treatment are fully discussed. In regard to malignant disease of the thyreoid, the author believes that sarcoma of the thyreoid is very uncommon, but he reports three cases of sarcoma in a series of twenty-one cases of malignant diseases of the gland. He advises the systematic removal of all thyreoid adenomata, even though the risk of malignant change may be no greater than 1%, as the best method to "eliminate malignant adenoma from its present position as the most common variety of malignant tumour of the gland".

The author strongly maintains for Parry the honour of first describing fully the clinical syndrome of exophthalmic goitre, and he has emphasized his point by publishing the portrait of Parry as a frontispiece. Parry's original paper describing his findings is abstracted. The last portion of the work deals with thyreotoxicosis; the author prefers to speak of primary and secondary thyreotoxicosis, rather than of exophthalmic goitre and toxic adenoma of Plummer.

The last section of the work is devoted to treatment. The author describes various points in his operative technique; he does not appear to be greatly impressed with the possibilities of X rays and radium in the treatment of exophthalmic goitre. He favours a combination of "twilight sleep", local anæsthesia and nitrous oxide and oxygen for his "bad risk" exophthalmic goitres.

At the end of each chapter a long list of references is appended. The author must be congratulated on the completion of a British monograph on thyreoid diseases which will be long referred to as a standard work by all interested in this subject.

ASTHMA.

"THE FRANCIS TREATMENT OF ASTHMA", by Alexander Francis, of London, expounds the theory that asthma and allied disorders have as their basis an instability of the vasomotor nerve mechanism, allergy, toxic, emotional and climatic factors acting as exciting agents setting up a bronchial vaso-dilation rather than the commonly believed bronchial spasm.[1]

The author has sought and claims to have found, as the rational basis of cure, a means of stabilizing the vasomotor mechanism by light cauterization of the nasal septum. Due consideration must be given to the exciting factors and to correct feeding, especially by reduction of the carbohydrate intake which if in excess is claimed particularly to upset the vasomotor balance. The avoidance of discovered irritants, intestinal detoxication, and the use of graduated and correctly timed exercises are advocated.

Prognosis is discussed. Strangely enough a healthy nose and a moderately raised blood pressure indicate a good outlook, while a low pressure and nasal

[1] "The Francis Treatment of Asthma", by A. Francis, M.B., B.Ch., B.A., M.R.C.S.; 1932. London: William Heinemann (Medical Books) Limited. Demy 8vo., pp. 160. Price: 7s. 6d. net.

polypi, especially in those sensitive to aspirin, are of bad portent. Quite the majority of some thousands of patients are claimed to be cured or materially benefited by this simple procedure; yet, in the minds of many practitioners, despairing so often of a cure, with the volume of knowledge at their disposal, the very simplicity of the method must engender scepticism. However, the fact that its exponent has, while also obviously giving consideration and trial to other views, for over twenty years remained convinced, should cause perusal of this book and test of this method by others and by rhinologists in particular, who must all at some time have seen relief given to an asthmatic patient from cauterization of an apparently diseased area of the *septum nasi*.

The text is clear and concise, the book of handy size for arm chair study, and there is a liberal series of case reports.

A TEXT BOOK FOR DENTAL SURGEONS.

SIR FRANK COLYER in collaboration with Dr. Evelyn Sprawson has produced the sixth edition of his text book "Dental Surgery and Pathology."[1] A great deal has been rewritten in this edition and several sections have been omitted. The student will find in the book accurate descriptions of diseases of the teeth and the oral tissues, but the practitioner who consults it on such subjects as the treatment of paradontal disease, the prevention of dental caries, or the relation of dental sepsis to systemic disease, will be disappointed.

Colyer's "Dental Surgery and Pathology" has been a standard work for a generation and is unequalled for its clear description of the diseases of the peridental membrane and oral mucous membrane, and classification and description of odontomes and tumours of the jaws. As a work of reference it is worthy of a place in the library.

SURGICAL PRACTICE.

THE first volume of "A Short Practice of Surgery", by Hamilton Bailey and R. J. McNeill Love, has just been published.[2] This volume deals principally with general surgical conditions, thyreoid, breast, mouth and tongue, the genito-urinary system, and bones and joints. It is a survey of general surgery, in which the essentials in surgical diagnosis and treatment are so clearly, concisely and vividly set out, that their ready assimilation by student readers is assured. Many apt diagrams and illuminating classifications, which have obviously been found useful by the authors in their long teaching experience, contribute greatly to this end. Much recent work, which has been generally accepted, has been included; treatment, previously accepted as sound, which experience has proved futile, has been omitted; thus, this selective treatment gives just that help which is needed by the medical student. Radium treatment of carcinoma of the breast is succinctly summarized as follows: "Radium is advocated by all in inoperable and recurrent cases. A few surgeons prefer radium to radical operation, but after a wave of enthusiasm in 1928 their numbers are declining. At the time of writing, operation, or operation *plus* radium, is in vogue throughout the world for the treatment of comparatively early mammary cancer."

[1] "Dental Surgery and Pathology", by J. F. Colyer, K.B.E., F.R.C.S., L.D.S., and Evelyn Sprawson, M.C., M.R.C.S., L.R.C.P., L.D.S.; Sixth Edition; 1931. London: Longmans, Green and Company. Demy 8vo., pp. 900, with illustrations. Price: 32s.

[2] "A Short Practice of Surgery", by H. Bailey, F.R.C.S., and R. J. McN. Love, M.S., F.R.C.S.; Volume I; 1932. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. 538, with 269 illustrations, of which 17 are coloured. Price: 20s. net.

Notable chapters are those on breast, kidney and ureters, and bladder and prostate. In the chapter on breast, commonly occurring conditions, such as retraction of the nipple, abnormal discharge from the nipple, suspected malignant disease, are dealt with in an attractive and original manner.

In dealing with tuberculous disease of the kidney, the art of the practised teacher is again apparent in the set of simple diagrams which are used to illustrate the various stages of its pathology. A scheme of the causes of hæmaturia will also appeal to the student mind.

The book is full of small excellently reproduced apt illustrations, many of which have been taken from the authors' own practices. It also contains many colour plates.

If any criticism is to be offered, it is that, in some instances, the authors have perhaps dealt with subjects too briefly, so that a previous knowledge would almost be necessary to the reader.

If the second volume follows the same lines as the first, and is up to its standard, the work will be appreciated by medical students and will have a successful life.

CANCER.

IN his book of nearly three hundred pages, written in a fine literary style, John Cope has set out to show that cancer is a racial degeneration effect.[1] He considers that the result is brought about largely by disuse of organs, most often sexual, though the pampering of the digestive tract by modern foods is operative in this region.

When he touches on the elimination of the unfit and the degenerate, his soundness will appeal to all readers; but no clear way of rapidly achieving this end is shown. Such a vital change as breeding only from the fit will have to come, if the author is right in his views. The change will necessarily be slow, but the sooner the whole world can be made to think about it, the better. The author has made his contribution to this end in this book which he gives to the lay public, as well as the professional. Though many things will be found in it that are a matter of opinion only, every chapter is interesting and will repay careful perusal from first to last.

PATHOLOGY FOR SURGEONS.

ANOTHER textbook of surgical pathology "for graduates and senior students" has been published.[2] The arrangement of material and the relative amount of space given to the various sections differ from those of similar textbooks. The authors write: "Presuming a certain knowledge of general pathology on the part of our readers, we have made only passing reference to such fundamental processes as inflammation, suppuration, ulceration and repair." This has allowed of more extensive treatment of some sections than would otherwise have been possible.

The book is of convenient size, the text is clear and the illustrations are uniformly good. The style is attractive, and there are very few typographical errors. A broad outlook on the subject of pathology is achieved in most departments, for example, in the introduction to the tumours, and, although this breadth of outlook is not sustained throughout all sections, the various viewpoints are succinctly stated. The literature, particularly recent material, has been closely

[1] "Cancer: Civilization: Degeneration", by J. Cope. London: H. K. Lewis and Company, Limited. Super royal 8vo., pp. 310, with 55 illustrations. Price: 15s. net.

[2] "Text-Book of Surgical Pathology", by C. F. W. Illingworth, M.D., F.R.C.S., and B. M. Dick, M.B., F.R.C.S.; 1932. London: J. and A. Churchill. Royal 8vo., pp. 677, with 290 illustrations.

followed, and the writers deserve congratulation on the way in which the latest views and the most recent work have been considered. The bibliographies have been carefully chosen and are, generally, those which would be most valuable to students pursuing the subject further.

In the 662 pages one cannot expect completeness. There are, however, a few unexpected omissions; for example, rhabdomyoma, which is being reported with increasing frequency, and the giant cells in Riedel's struma. The section on *osteitis fibrosa* is disappointing. An occasional error creeps in; for example, the statement about the onset of gangrene in relationship to arterio-sclerotic change in diabetes. On the other hand, the book has good points which are absent from similar works, such as the deletion of some of the older and unsatisfactory terms in the section on the sarcomata.

Altogether the book is excellent for the purpose for which it was written; it bears the stamp of the Edinburgh school, and is, we think, one of the best works available at present on surgical pathology.

ANÆSTHESIA.

JAMES G. POE'S book on general anæsthesia, which has just reached its second edition, contains several features of interest.[1] There is a useful chart in which the various signs to be elicited in the stage of narcosis of ether anæsthesia are graphically set out; at the end of each chapter there is a brief, tabulated summary by means of which the reader can rapidly refresh his memory of the subject matter. These features, of course, are of more value to the student than to the practitioner of medicine.

The book contains a great deal of information in comparatively few pages; but the space allotted to various subjects is not always proportionate to their relative importance. For example, the "open" ether method is given four and a half pages only, and is quite inadequately described. Nitrous oxide anæsthesia and ethylene anæsthesia are discussed far more fully.

The so-called basal anæsthetics are briefly dealt with, and a guarded opinion on their value is given. The section dealing with spinal anæsthesia may be regarded as adequate when the size of the book is considered; the discussion of local and regional anæsthesia is valueless to any person desiring a knowledge of the technique of these methods.

The author apparently desires to mention everything in the world of anæsthetics. His book would be of greater value if he mentioned a little less and described a little more.

THE ABDOMINAL CAVITY AND PERITONEUM.

UNDER the title: "A Clinical Study of the Abdominal Cavity and Peritoneum", Edward M. Livingston, of Bellevue Hospital, New York, presents for the approval of the medical reader a book of 800 pages.[2] In this first volume he deals with the empty abdominal cavity and the gastro-intestinal tract, while he promises a further volume dealing with the solid abdominal organs and the peritoneal membrane. After study of the volume under review the appearance of the second volume will be eagerly awaited.

The book is well printed and contains over 300 well chosen illustrations and a list of nearly 400 references to original articles.

[1] "Modern General Anæsthesia: A Practical Handbook", by J. G. Poe, M.D.; Second Edition; 1932. Philadelphia: F. A. Davis Company. Demy 8vo., pp. 231, with illustrations. Price: $2.50 net.

[2] "A Clinical Study of the Abdominal Cavity and Peritoneum", by E. M. Livingston, B.Sc., M.D.; 1932. New York: Paul B. Hoeber. Imperial 8vo., pp. 888, with 371 illustrations.

A feature which adds much to the interest and pleasure of the reader is the inclusion of a group of eponyms of those whose names are inseparably linked with the advancement of our knowledge of the abdomen and of excerpts from their original articles.

The subject matter is considered under three main subdivisions. In the first, dealing with the empty cavity, the author discusses the embryology and anatomy of the roof, floor and walls of the abdominal cavity with their associated herniæ, methods of abdominal topography and clinical examination. The author describes intraabdominal drainage areas and the effects of intraperitoneal fluids and pressures. In the second section the embryology, the distribution and final attachments of the gastro-intestinal tract and its anatomical and physiological divisions are discussed in relation to their arterial and lymphatic supply.

All through these two sections of the book there is close correlation between anatomical, physiological, pathological and clinical aspects of the abdomen, and it is the author's aim to explain one in terms of the other. Many facts not stressed in the usual textbook are discussed and explained, and many generally accepted as uninteresting and barren are presented in a new light and endowed with fresh importance.

It is in the third section, which deals with visceral neurology, that the author presents the results of his investigation into the nature and significance of abdominal pain. He first studies the innovation of the abdominal wall and of the gastro-intestinal tract with its sympathetic and parasympathetic subdivisions, the clinical significance of metamerism, viscerogenic reflexes and their interpretation. He stresses and strongly advocates the value of the adequate stimulus in the demonstration of hyperalgesia and of visceral maximal points, and the usefulness of arbitrary topographical boundaries of hyperalgesia in the differential diagnosis of abdominal pain.

The work was attempted as a help to post-graduate study. It has certainly been successful, and within its covers students, surgeons and physicians will find much to interest and enlighten them.

EXERCISE.

THE book on the "Physiology of Exercise", by Ferdinand August Schmidt and Wolfgang Kohlrausch, contains much valuable information, and should be read by all medical practitioners interested especially in athletics and physical culture.[1]

The authors have not succeeded in making the book easily readable. Perhaps it has suffered in the translation, in which no less than three people have been concerned. Its dulness may be an illustration of the truth of the ancient adage concerning the number of cooks and the quality of the broth.

Obviously the authors have made an earnest study of their subject. Their various theories are interesting and the results of many of their observations are important.

[1] "Physiology of Exercise", by F. A. Schmidt, M.D., and W. Kohlrausch, M.D., translated by C. B. Sputh, M.D.; 1931. Philadelphia: F. A. Davis Company. Royal 8vo., pp. 216, with 44 engravings. Price: $2.50 net.

Proceedings of the Royal Australasian College of Surgeons

A MEETING OF THE COUNCIL OF THE ROYAL AUSTRALASIAN COLLEGE OF SURGEONS was held at the British Medical Association Building, 135, Macquarie Street, Sydney, on September 3, 1932.

The Council expressed sympathy with Alan Newton, who was unable to attend owing to illness.

Appointment of Honorary Assistant Secretary.

Acting under the authority given to it by the Council at its meeting on August 6, 1928, the Executive Committee had appointed Julian Smith, Junior, as Honorary Assistant Secretary of the College. This appointment was approved by the Council.

Election of Fellows.

The following Fellows were elected:

New South Wales: C. L. Chapman, H. M. de Burgh, J. G. W. Hill, K. L. H. Kirkland, S. F. Lynch, E. McA. Steel, J. Grenville Waine and C. E. Winston.

New Zealand: W. M. Brown, E. Ewart Brown, W. F. Buist, J. R. Connolly, H. K. Corkill, E. H. M. Luke, W. J. MacDonald, R. B. Martin, E. R. Reay and W. S. Robertson.

Queensland: Harold Crawford, G. F. Meade and H. W. Savige.

South Australia: Brian F. Moore.

Victoria: J. G. Winter Ashton, E. G. Dermer and A. M. Lazarus.

The following Junior Members were elected:

New South Wales: E. J. Egan.

South Australia: Ian A. Hamilton.

Address to the Council of the Royal College of Surgeons of England.

The following address was conveyed personally by the President to the Council of the Royal College of Surgeons of England:

> The President and Council of the Royal Australasian College of Surgeons have gratefully received from Mr. C. H. Fagge the message conveying the sympathy and goodwill of the President and Council of the Royal College of Surgeons of England.
>
> This message and the visit to Australia of Mr. C. H. Fagge, will encourage the Fellows of the Royal Australasian College of Surgeons in their endeavours to advance the Science and Art of Surgery for the common weal.
>
> The interest and friendship evinced by the Royal College of Surgeons of England in the foundation and development of the Royal Australasian College of Surgeons will strengthen the feelings of brotherly goodwill in our profession throughout the Empire.
>
> H. S. NEWLAND, President.
> R. HAMILTON RUSSELL, Censor-in-Chief.
> ALAN NEWTON, Honorary Secretary.

A letter from the President was read relating his sympathetic reception by the Lord President and Council of the Royal College of Surgeons of England, who asked him personally to convey to the Council and Fellows of this College their warmest greetings and good wishes.

Senior Surgical Qualifications.

The Council had previously defined a senior surgical qualification as one containing a primary examination in anatomy and physiology and a final examination in surgery, surgical anatomy and surgical pathology. With a view to defining more precisely the term "primary examination", the following resolution was passed:

> That, as this College was instrumental in obtaining uniformity in senior surgical qualifications in the Australian Universities, it believes that it must insist on a standard similar to that demanded for these qualifications. In other words, the standard of the primary examination must be equivalent to that of the M.S. degrees in the Australian Universities.

The Council, at its first meeting on February 5, 1927, gave notice that, after a period of five years, the possession of a senior surgical qualification would be required of candidates seeking admission to Fellowship. The term "senior surgical qualification", however, had not been exactly defined until March, 1931, and during the time which elapsed after the Council gave notice of its intention to insist upon a senior surgical qualification, it was evident that there were some men who had obtained qualifications which they thought would entitle them for consideration to Fellowship. It was thought, therefore, that candidates who had obtained, within this period of five years, any senior surgical qualification, should be considered apart from the Council's ruling of March, 1931, and the following resolution was passed:

> That the operation of regulation 5, section 7, in the Handbook, 1931, be suspended in the case of candidates possessing a senior surgical qualification without a primary examination, provided that those candidates obtained the qualification between February 5, 1927, and February 5, 1932.

Permanent Headquarters.

The Council approved of plans for a Permanent Headquarters Building. These plans show a splendid façade, as seen in the accompanying illustration. The elevation shows a very dignified entrance porch. In the scheme adopted, the main floor (ground floor) provides a large foyer, a lecture room to seat 280 persons, a room for Fellows or for Council meetings, executive offices, and a large library. As the site is a sloping one, a large room, 40 feet by 50 feet (which is not a basement), is very economically incorporated. This room will seat 225 persons, or will serve the purpose of a museum of clinical or surgical pathology. The second story, the interior of which is left unfinished in order to save expense, can be utilized at any time for research rooms or for any contingencies which may occur in the future. In this plan, the two-storied aspect, the size of the building, and the character of the façade, lend a dignity and importance which are in keeping with the important buildings in the neighbourhood and with the site on which it stands. This scheme not only provides for the present requirements of the College, but gives some potentiality for future needs.

Library for Headquarters.

In the new building, which it is proposed to erect as a headquarters for the College, provision will be made for a library and for research in surgical literature. With this end in view, the Editorial Committee of THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY has been, for some time, accumulating surgical journals from other countries, which it has received in exchange for its own

Façade of proposed Permanent Headquarters Building for the Royal Australasian College of Surgeons.

Australasian Journal. The Executive of the College now feels that the time is opportune to begin adding to this nucleus of a library, in order that, when the building is completed, enough books and surgical literature will have been accumulated to form a small library. It would, therefore, appeal to Fellows for gifts of books, reprints, back numbers of surgical journals, or for any literature or pictures of historical interest, which would be of value in establishing such a library.

Communications in relation to this project may be addressed to the Librarian, Mr. G. R. A. Syme, 6, Collins Street, Melbourne.

Corrigenda.

REPORT OF ANNUAL MEETING, 1932.

MR. C. H. FAGGE writes that several mistakes of fact appeared in the issue of March, 1932, in the report of Sir Henry Newland's speech, made at the Annual Meeting, 1932. Mr. Fagge writes:

> I am referred to twice on page 444 as Charles Hilton Fagge, whereas my name is Charles Herbert, given correctly on page 445. John Hilton was my great uncle and Charles Hilton Fagge was my uncle, not my father.

The mistakes were unfortunate and were due to the inadvertent use of an uncorrected copy of Sir Henry Newland's speech. The statements made by Sir Henry Newland at the meeting were correct.

FIBROCYSTIC EPITHELIOMA OF THE JAW.

H. R. DEW and D. I. Miller who published an article in the March, 1932, issue of this journal entitled "Fibrocystic Epithelioma of the Jaw", write that they have received a letter, dated June 9, 1932, from Dr. H. R. Sear in which he refers to their statement on page 363:

> *Radiography*: The radiogram shows typical appearances with polycystic absorption and expansion of the mandible. There will be no evidence of an unerupted tooth.

Dr. Sear stated that this view was quite opposed to that of S. O. Goodsell "Polycystic Adamantine Epithelioma", *International Journal of Orthodontia*. Volume XVII, March, 1931, who wrote as follows: "All the reported cases of adamantinomas which I reviewed contained unerupted impacted third molars". He added that the article by Goodsell figured among the references given by Dew and Miller.

H. R. Dew and D. I. Miller write under date, July 6, 1932, as follows: "In order to emphasize the contrast between this tumour and the commoner well known odontomata, we stated that in the former there will be no evidence of an unerupted tooth, which is a constant finding in the latter.

"It must be admitted of course—there have been several such cases reported—that there may be coincident non-eruption of a tooth in the disease under question. This is particularly prone to occur apparently in the region of the third molar, so that the presence of a non-erupted tooth particularly in this situation, must not be taken to exclude fibrocystic epithelioma."